A STUDY ON

"CLINICAL AND ENDOSCOPIC PREDICTORS OF THE OUTCOME OF CORROSIVE INGESTION"

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CERTIFICATE

This is to certify that the dissertation entitled "**CLINICAL AND ENDOSCOPIC PREDICTORS OF THE OUTCOME OF CORROSIVE INGESTION**" is a bonafide original work of Dr. KARTIKAYAN. R. K., in partial fulfilment of the requirements for M.D. Branch– I (General Medicine) Examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in APRIL 2014 under my guidance and supervision during the period of June 2013 - December 2013.

PROF. DR. E. DHANDAPANI, M.D.,
Professor of Internal Medicine,
Guide and supervisor
Institute of Internal Medicine
Madras Medical College
Rajiv Gandhi Government
General hospital, Chennai – 600003.

PROF. DR. K. SIVASUBRAMANIAM, M.D.,
Director and Professor
Institute of Internal Medicine
Madras Medical College
Rajiv Gandhi Government General Hospital,
Chennai – 600003.

Dr. V.KANAGASABAI, M.D., M.B.A.,
Dean
Madras Medical College &
Rajiv Gandhi Government
General Hospital,
Chennai-3

DECLARATION

I hereby solemnly declare that this dissertation entitled "**CLINICAL AND ENDOSCOPIC PREDICTORS OF THE OUTCOME OF CORROSIVE INGESTION**" was done by me at Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai-3 during June 2013- December 2013 under the guidance and supervision of Prof. Dr. E. DHANDAPANI, M.D. This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfilment of requirement for the award of M.D. Degree Branch I (General Medicine).

Place: SIGNATURE OF THE CANDIDATE

Date:

DR. KARTIKAYAN. R.K.
MD GENERAL MEDICINE,
Post Graduate Student,
Institute of Internal Medicine,
Madras Medical College,
Chennai – 600003.

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ABBREVIATIONS

ABG	-	Arterial Blood Gas
aPTT	-	activated Partial Thromboplastin Time
ATN	-	Acute Tubular Necrosis
BAPN	-	β -Amino Proprio Nitrile
CAPE	-	Caffeic Acid Phenethyl Ester
CNS	-	Central Nervous System
CT	-	Computed Tomography
DIC	-	Disseminated Intravascular Coagulation
ECG	-	Electrocardiogram
EGD	-	Esophago Gastro Duodeno scopy
GERD	-	Gastro Esophageal Reflux Disease
GI	-	Gastro Intestinal
IV	-	Intra Venous
LDH	-	Lactate Dehydrogenase
NAC	-	N – Acetyl Cysteine
NPO	-	Nil Per Oral
pH	-	negative logarithm of Hydrogen ion concentration
pKa	-	dissociation constant of acid/alkali
PPI	-	Proton Pump Inhibitors

PT	-	Prothrombin Time
TAR	-	Titrable Acid/Alkali Reserve
Tc99m	-	Technitium 99 metastable
TPN	-	Total Parenteral Nutrition
USG	-	Ultrasonography

Clinical and endoscopic predictors of the outcome of corrosive ingestion

Abstract

Corrosives are common substances to be ingested either with a suicidal intent or accidentally. Following ingestion they cause a wide spectrum of injury to the GI tract both acute and delayed. The consequences are associated with significant mortality and morbidity.

The aim of this study was to identify the factors in a given patient with history of corrosive ingestion the predictors of significant corrosive injury and also to predict the long term outcome.

Key words: “corrosives”, “esophageal and gastric burns”, “strictures”, “Upper GI endoscopy”

Study

50 patients with history of corrosive ingestion who presented within 24 hours of consumption and in whom the initial upper GI endoscopy was done within 24 hours of consumption were taken as the study after excluding complications such as respiratory distress and perforation. Detailed history regarding the consumption and symptoms was taken and thorough physical examination was done. They all underwent initial upper GI endoscopy and their injuries were categorized and managed accordingly.

They were serially followed and a follow-up endoscopy was done at 6 weeks to look for the extent of healing and the presence of strictures.

The outcome was analysed statistically based on several parameters and the results were obtained.

Corrosive ingestion was more common in males and in the age group of 20 – 30 years. Acids were more commonly consumed than alkalis in our study. Suicidal intention was the most common circumstance of poisoning and it was associated with significant injury along with accidental consumption under the influence of alcohol where again the incidence of injury was higher. Consumption of more than 50 ml was associated with significant injury. Symptoms and signs (oropharyngeal burns) were not reliable in predicting the injury.

The extent of the lesions on initial endoscopy had the highest correlation in predicting the occurrence of strictures with higher grade lesions having the most significant association. Placement of a NG tube for providing nutrition did not serve the purpose of reducing the occurrence of strictures in patients with significant injury.

Conclusion:

While poisoning with corrosive substances is a common occurrence in our part of the country, knowledge is required about the predictors of higher grade lesions and long term sequelae. The factors in our study which were associated significant injury as well as long term sequelae are consumption of more than 50 ml, suicidal ingestion or accidental ingestion under the influence of alcohol, higher grade lesions at initial endoscopy.

INTRODUCTION

INTRODUCTION

"All substances are poisons; there is none which is not a poison. The right dose differentiates a poison from a remedy."

Paracelsus

Toxicology is the study of adverse effects of xenobiotics on human beings. Modern toxicology goes beyond the study of adverse effects of exogeneous agents to the study of molecular biology, using toxicants as tools.

Corrosive substances are common household substances that can be ingested either accidentally or intentionally with suicidal intent. Ingestion of corrosive chemicals causes a wide spectrum of injury to the Upper gastrointestinal tract that may be moderate or fatal and may lead to lifelong handicap[1]. Hence patients who present with the history of having consumed corrosive substances should be evaluated in an emergency basis not only to identify early complications such as perforation and haemorrhage but also to take care of the nutrition of the patient and to assess the risk of delayed damages such as stricture, etc.

Corrosives substances may be broadly classified into acids and alkalis. Acids and alkalis produce damage to the mucosa of the gastrointestinal tract by means of coagulative necrosis and liquefactive necrosis respectively.

The initial investigative modality of choice for evaluating the extent of injury in patients with history of consumption of corrosives is Upper GI endoscopy. The ideal time for an Upper GI endoscopy in patients who have consumed corrosives is usually in the initial 12 – 24 hrs. An initial Upper GI endoscopy gives us useful data on the location, severity and extent of the post-corrosive injuries and the endoscopic classification is of substantial importance for establishing the diagnosis[1].

The most important predictor of the occurrence of complications in a patient who has consumed corrosives is the extent of tissue injury that has occurred which can be assessed by an Upper GI endoscopy. The extent of injury in turn is influenced by several factors such as the type of substance consumed, amount and concentration of the substance, duration since consumption and the act of swallowing[1].

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES

1. To evaluate 50 cases of corrosive injury of GI tract
2. To analyse the clinical profile of 50 cases of corrosive injury of GI tract.
3. To analyse the outcome of 50 cases of corrosive injury of GI tract.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Definitions

Corrosive substances

A corrosive is a substance that causes damage on contact with tissue surfaces both histologically and functionally. Corrosive substances can be classified typically into two types based on their pH and proton donating/accepting nature.

- Acids are substances which act as proton donator and usually have a pH below 3.
- Alkalis are substances which act as proton acceptors and usually have a pH above 11.

When corrosive substances come in contact with tissues, there is release of thermal energy for neutralising the corrosive. This thermal energy is responsible for the damage caused to the tissues

There are several factors which influence the extent of injury caused by the corrosive to the GI tract like the volume of the substance consumed, pH of the corrosive, concentration at which it was consumed, ability of the

substance to penetrate tissues and a property of the corrosive known as titrable acid/alkali reserve (TAR). TAR is a quantification of the amount of neutralizing xenobiotic needed to bring the pH of a corrosive to that of physiologic tissues. The ability of the substance to cause tissue damage is usually directly proportional to the TAR of the substance.

Corrosives are present in many household items that are used in day-to-day practice. They are available in both solid and liquid forms, with variations in viscosity, concentration of solution, and pH. The circumstance of consumption of corrosive agent is usually with suicidal intent in adults. Accidental unintentional exposure can occur in children or in adults who are under the influence of alcohol or in psychiatric patients.

Table 1 - List of alkalis and their common applications.

Chemical	Applications used as
Sodium and potassium hydroxide (lye)	Detergents and washing powders, paint removers, drain cleaners, oven cleaners, denture cleanser
Sodium hypochlorite	Bleaches, cleansers
Sodium borates, carbonates, phosphates and silicates.	Detergents, electric dishwashing preparations, water softeners, purex bleach
Ammonia (ammonium hydroxide)	Toilet bowl cleaners, metal cleaners and polishers, hair dyes, jewellery cleaners.
Potassium permanganate	Illegitimate abortifacient
Phosphorus	Matches, rodenticides, insecticides, fireworks
Benzalkonium chloride	Detergents, floor and toilet bowl cleaners.
Mercuric chloride ($HgCl_2$)	Preservative

Table 2 – List of acids and their common applications

Chemical	Applications used as
Hydrochloric acid	Metal cleaners, toilet bowl cleaners (5 – 25%), swimming pool cleaners
Sulfuric acid	Battery acid (25 – 30%), drain cleaners (95 – 99%), toilet bowl cleaners (8 – 10%)
Sodium bisulfite	Toilet bowl cleaners
Oxalic acid	Disinfectant, furniture polish, rust and stain remover
Hydrofluoric acid	Anti rust products
Formaldehyde (formic acid)	Deodorizing tablets, plastic members, fumigants, embalming agents (60%)
Carbolic acid (phenol)	Antiseptic, preservatives
Acetic acid	Permanent wave neutralizers, photographic stop bath (6 – 40%)
Boric acid	Roach powders, water softeners, germicide
Selenious acid	Gun bluing agent

Pathophysiology of injury following corrosive ingestion

Alkali ingestion

The primary pathology that occurs in the tissues following alkali exposure is liquefaction necrosis. The basic mechanism is the formation of hydroxide ions from the alkali once they come in contact with the tissues. The entire process includes protein dissolution, collagen destruction, fat saponification, cell membrane emulsification, transmural thrombosis, and cell death. Vascular thrombosis occurs following the necrosis.

In case of alkali ingestion, the site most commonly affected is the esophagus. The stomach is relatively spared of the damage of neutralization by acid; with few patients having damage in the small intestine as well[3]. Acutely, injury can range from erythema and edema, through erosion and ulceration, to necrosis and perforation. The initial alkali injury hence can be transmural and if associated with perforation can lead to mediastinitis, and peritonitis[2]. External sloughing and ulceration occur a few days after ingestion. In animal models of alkali injury, the tensile strength of the esophagus is lowest from day 3 to day 14, a vulnerable period for esophageal perforation. Finally, extensive granulation tissue, fibroblastic activity, and collagen deposition occur over weeks, leading to chronic stricture formation. Although collagen organization and

epithelial repair may continue for months, severely affected segments have permanent shortening, dysmotility or atony, and stricture. Finally, the incidence of squamous cell carcinoma at the sites of strictures in the esophagus increases 20 to 40 times after alkali injury, with a latency period of decades.

Alkali solutions have the hazard of being consumed in larger quantities, even when consumed accidentally, by virtue of being odorless and tasteless. Moreover unlike with acids, there is little or no immediate pain to deter an accidental ingestion and hence it is well recognized that even an accidental ingestion of a small amount of concentrated alkali can result in significant injury.

The factors that influence the severity of alkali injury include concentration, volume, pKa, pH, TAR, formulation (solid vs. liquid), and viscosity of the alkali, as well as transit time, pre-existing contents (food, secretions), and premorbid condition of the gastrointestinal tract. Solid or granular alkali tends to cause localized injury, especially at sites of anatomic narrowing, whereas liquid alkali causes more diffuse, circumferential injuries. Intentional ingestions generally involve larger volumes and can cause burns distally into the duodenum[4].

Acid ingestion

Acids induce tissue injury by means of tissue protein desiccation to produce coagulation necrosis in contrast to liquefactive necrosis caused by alkali ingestion. This occurs by a process in which the dissociated protons (H^+) from the ingested acid, after hydration with H_2O obtained from the cells form hydronium ions ($H3O^+$), results in cellular protein desiccation, denaturation, and precipitation. This process of protein precipitation results in eschar formation and hence the acid-induced damage is usually limited to the more superficial layers of mucosal tissue as penetration into the deeper layers is impeded by the presence of the eschar. Even though this eschar is postulated to be protective, acid ingestion may induce full-thickness burns secondary to tissue sloughing, with resultant esophageal or gastric perforation and even potentially fatal complications.

Caustic-induced injury to the tissues can be generally characterized by three phases. First is the inflammatory phase (which lasts for about 4 – 7 days) in which there are thrombotic events in the vasculature with cell necrosis eventually leading to the destruction of the columnar epithelium of the mucosa and the submucosa. Generally, at 72 to 96 hours after ingestion an ulcer develops after superficial mucosal necrosis and sloughing. The second phase, the high-risk time for perforation to occur,

begins around 3 days and lasts up to 2 weeks after ingestion. Attempts to repair the necrotic areas initially by filling them with granulation tissue and subsequently with collagen occur at this time. Lastly, if the gastrointestinal mucosa has sustained a severe caustic-induced injury, an excessive amount of fibrous tissue may form, resulting in stricture formation 2 or more weeks after ingestion.

The pattern of injury of the gastrointestinal tract following is also different from that following alkali ingestion, the most common pattern being concomitant involvement of the esophagus and stomach[5]. In rare occasions, the esophagus is spared of damage with severe damage noted in the stomach and is probably related to the rapid transit time of liquid acids through the upper gastrointestinal tract[6]. In stomach the injuries are common in the antrum. The reason for the predilection to affect the antrum is due to the “magenstrasse” flow of liquid acids along the lesser curvature of the stomach with resultant pooling in the pylorus secondary to acid-induced pylorospasm. Skip lesions from acid ingestions may be a function of viscosity and contact time. The relative sparing of the duodenum may be due to the pylorospasm and the alkaline pH of the duodenum, but injury does occur[7].

Another important feature is that acids when ingested, the amount ingested is likely to be low when compared to alkali due to the offensive smell and immediate pain following ingestion. Thus in certain cases acid ingestion causes less overall damage when compared to alkalis.

Clinical presentation

Clinical presentation in a patient who has consumed corrosive can range from being entirely asymptomatic to being extremely moribund. The common symptoms include

- Pain which can be at multiple sites such as oropharyngeal pain, chest pain, epigastric or abdominal pain
- Burns in the oral cavity and oropharynx - Examination of this region may be unremarkable or reveal burns ranging from erythematous mucosa to mucosal erosions with pseudomembrane formation to actual necrosis of the buccal mucosa and uvula. The absence of burns in the oropharyngeal region after the ingestion of corrosive does not preclude the presence of esophageal or gastric injury[8].
- Nausea, vomiting, dysphagia, refusal to swallow and drooling of secretions
- About 40% patients are asymptomatic with normal physical examination.

The important symptoms that should arouse the suspicion of complications include

- Haematemesis or melena indicates upper gastrointestinal bleeding which could be due to the corrosive injury induced burns per se, due to perforation or due to great vessel erosion.
- Respiratory distress if present may be due to
    - aspiration of contents (especially when the patient has history of vomiting),
    - esophageal perforation (when present with persistent retrosternal pain, fever, subcutaneous emphysema over the neck and chest, Hamman's mediastinal crunch) or
    - due to corrosive induced laryngeal edema (which manifests as hoarseness, stridor and aphonia)
- Perforation of subdiaphragmatic viscera is rare and may present with fever, abdominal guarding and rigidity with ileus.
- Acid can readily be absorbed across the gastric mucosa leading to a more severe systemic acidemia. Depending on the acid ingested, an anion gap (e.g., sulfuric acid) or non–anion gap (hydrochloric acid) metabolic acidosis may result.

- Rarely in patients who present late may show signs of end stage complications like shock, metabolic acidosis, DIC, signs of vital organ hypoperfusion

Certain corrosives have specific systemic toxicities when absorbed into the circulation[9].

Table 3 – systemic effects of specific corrosive agents[35]

Corrosive Agent	Systemic Symptoms
Formaldehyde	Metabolic acidosis; formate poisoning
Hydrofluoric acid	Hypocalcemia; hyperkalemia
Methylene chloride	CNS depression; cardiac arrhythmias; converted to carbon monoxide
Oxalic acid	Hypocalcemia; renal failure
Paraquat	Pulmonary fibrosis
Permanganate	Methemoglobinemia
Phenol	Seizures; coma; hepatic and renal damage (ATN – olive green urine) Respiratory depression, pulmonary edema
Phosphorus	Hepatic and renal injury (see Phosphorus)
Picric acid	Renal injury
Silver nitrate	Methemoglobinemia
Tannic acid	Hepatic injury

Delayed complications:

Those patients surviving a few weeks after a grade II or III injury may subsequently present with dysphagia and vomiting from stricture

formation. Strictures may also present with esophageal motility disorders caused by impaired smooth muscle reactivity[23].

Other complications reported include motility abnormalities of the pharynx and esophagus, formation of aorto- and tracheoesophageal fistulas, delayed massive hemorrhage from erosion into a great vessel, and pulmonary thrombosis[33].

Another dreaded long term complication is the association of malignant potential in patients with strictures following alkali ingestion[34]. Long-term survivors of moderate and severe injury of the esophagus have a risk of esophageal carcinoma (squamous cell type) that is 1000 times higher than that of the general population and appears to present with a latency of up to 40 years.

Approach to a patient with history of Corrosive ingestion

History

- Name, concentration, and amount of acid/alkali ingested
- Time of ingestion
- Accidental (small volumes) vs. suicidal (large volumes)
- Vomiting after ingestion (aspiration risk)
- Presence of food in the stomach prior to ingestion

Signs and symptoms of organ damage

- Pain – oropharyngeal, retrosternal, epigastric
- Respiratory distress, stridor, hoarsensess of voice
- Oral and/or oropharyngeal burns
- Dysphagia, drooling, vomiting
- Haematemesis or melena
- Fever
- Shock, DIC, Metabolic acidosis

Figure 1 – Picture showing burns in the lips and tongue of a patient with alkali ingestion (sodium hydroxide 100%).

Table 4 - Differential diagnosis of patients with history of corrosive ingestion

Airway signs and symptoms Infections (epiglottitis, croup, deep neck space infections) Foreign body Anaphylaxis/Angioneurotic edema Thermal burns Asthma/Bronchiolitis/Croup	Esophageal obstruction Foreign body Malignancy Dysmotility Achalasia
Upper GI bleeding Trauma Peptic ulcer disease Esophageal varices Malignancy Gastritis Mallory-Weiss tear	Mediastinitis Trauma (Boerhaave syndrome, iatrogenic, penetrating trauma) Malignancy
Shock – septic, cardiogenic (especially in elderly)	

Investigations

All patients with history of corrosive ingestion should be subjected to routine blood investigation such as Complete blood counts, blood grouping, renal and liver function test, coagulation parameters, ECG and ABG. Elevated Prothrombin time (PT) and activated Partial Thromboplastin time (aPTT) and an arterial pH below 7.22[10] are suggestive of DIC and metabolic acidosis indicating severe nature of corrosive injury. Changes in

the levels of Uric acid (increase), phosphate and alkaline phosphatase (both decrease) were found to be useful in predicting esophageal injuries in children in one prospective study [11].

Radiography

X rays of the chest and abdominal in the initial stages may not be useful in grading the severity of injury directly. But they are nevertheless indicated for detecting gross signs of esophageal or gastric perforation which include pneumomediastinum, pneumoperitoneum, and pleural effusion (any of which would be an indication for emergency surgical management) and also to rule out aspiration. The major disadvantage of, these studies however is their limited sensitivity, and hence an absence of findings cannot be taken as an evidence of the absence of perforation[33].

An X-ray Chest PA view is best to visualize intraperitoneal although occasionally a pneumoperitoneum may be seen only in a lateral chest X ray. Free intraperitoneal air adjacent to the liver demonstrated in lateral abdominal radiographs are also suggestive of perforation and may be used in patients who are too ill for an upright chest X ray. In patients with significant corrosive ingestion where there is high risk of perforation, CT is more sensitive X rays for detecting hollow viscus perforation[12].

Contrast studies – A contrast esophagram is useful for defining the extent of esophageal injury in both in the acute as well as in the delayed phases. An enteric contrast study (esophagram and upper GI series) can be obtained 24 hours after the ingestion especially in patients for whom there is a high suspicion for esophageal perforation in whom adequate visualization of the upper gastrointestinal tract by endoscopy is not possible due to the extensive nature of injury (as in grade IIb circumferential burns or grade III burns). The presence of perforation is indicated by extravasation of contrast outside the GI tract[33]. Late after the ingestion contrast studies are useful for detecting stricture formation and also for assessing esophageal motility.

The choice of contrast agent to be used is also to be considered before taking up the contrast study as both Barium contrast and water soluble contrast have their pros and cons[33,34]. Water-soluble contrast would be ideal when perforation is suspected as it is has less chance of causing irritation to mediastinal and peritoneal tissues even if extravasated. However, the resulting study with water soluble contrast is suboptimal because of the fact that barium contrast agents are more radiopaque than water-soluble agents and have the advantage of offering greater radiographic detail[13]. A staged approach is also sometimes employed, with

barium being administered only after a negative study using water-soluble contrast medium. In addition, if there is risk of aspiration, barium is preferred because water-soluble contrast material can cause a severe chemical pneumonitis. The findings on contrast studies that should arouse a suspicion of perforation include esophageal dilation, displacement of the pleural reflection, and widening of the pleuroesophageal –line[33].

Figure 2 – Barium swallow in a patient with alkali ingestion showing atonicity and poor coating of the esophagus, suggesting edema and intramural penetration.

Figure 3 – Repeat Barium swallow in the same patient showing mid-esophageal stricture with sparse passage of barium beyond the stricture and thinning of the distal portion of the esophagus.

Upper GI endoscopy:

As the signs and symptoms do not match the degree of injury, an upper endoscopy examination should be performed in the first 24 to 48 hours in all patients with history of corrosive ingestion who do not have any clinical or radiographic evidence of perforation[14]. The usefulness of a promptly performed early upper GI endoscopy lies not only in diagnosis of injury and

grading the extent of injury but also in assessing the long term prognosis and in guiding therapy. Other well recognized advantages of an early upper GI endoscopy include shortening of time that the patient has to forego nutritional support, permitting more precise therapeutic regimens and also in early discharge of patients with normal findings or minimal evidence of GI tract injury[15]. However endoscopic examination may be normal with no evidence of injury in as many as 40 – 80% of the patients with history of corrosive ingestion.

Indications

- All symptomatic patients
- All Young Children even if asymptomatic.
- All Patients who have intentionally ingested

Endoscopy may be omitted in patients with history of accidental ingestion of small quantity with no symptoms or signs and symptoms described above. An upper GI endoscopy if performed in such cases would be to document absence of injury in the GI tract

Contra indications

- Obvious signs of a severe full-thickness injury

- Esophageal or gastric necrosis with pleural irritation
- Mediastinal irritation
- Peritonitis
- Free air in the abdomen

The ideal time for performing an endoscopy in a patient who has consumed corrosive would be in the 1st 24 hours following ingestion. It may be done up to 48 – 72 hours following ingestion but should not be done between 5 days and 2 weeks post-ingestion as it is at this time that wound strength is least and the risk of perforation is greatest. A delay of 4 to 6 hours before initial endoscopy is recommended to avoid underestimating the severity of injury[16].

If upper airway edema due to burns is known or suspected, an endotracheal tube should be placed prior to esophagoscopy. However, esophagoscopy should not be deferred until resolution of upper airway swelling. Previous recommendations to abandon the endoscopic procedure at the level of the first full-thickness or circumferential burn originated in the era of rigid endoscopy. The comfort and experience of the endoscopist mainly decides the choice between a rigid and a flexible endoscope[33]. The flexible endoscope in its part has certain advantages like being of a smaller

diameter but however requires gentle insufflation of air for achieving better visualization. A prospective evaluation of the role of fiberoptic endoscopy in the management of corrosive ingestions recommended the following guidelines:

a. direct visualization of the esophagus prior to advancing the instrument,
b. minimal insufflation of air,
c. passage into the stomach unless there is a severe (particularly circumferential) esophageal burn, and (a rule if violated can incresase the risk of endoscopy associated perforation)
d. Avoidance of retroversion or retroflexion of the instrument within the esophagus.

Hence in a patient who is hemodynamically stable with no evidence of perforation, an earnest attempt should be made to visualize the esophagus, stomach and duodenum after a corrosive ingestion regardless of the signs and symptoms.

The major pitfall in endoscopy is the limited evaluation of gastrointestinal injury, as only the mucosa is visualized and evaluated sparing the serosal side. Ulcers in the stomach are particularly susceptible to this pitfall as they which may appear necrotic and black which may be a result of a true burn

through the layers of the stomach, or due to the effect of stomach acid on the blood exposed from a shallow lesion. To overcome these issues the use of endosonography has shown to improve the assessment of depth of injury in recent studies[17]. Often definitive evaluation is possible only with laparoscopy or laparotomy which allows the serosal as well as the mucosal surfaces to be visualized directly.

The risk of perforation following endoscopy is usually associated with advancement of the endoscope beyond severe circumferential lesions or with the use of rigid endoscope in a child or in an un-cooperative patient[34].

Endoscopic grading of Corrosive injury to the GI tract

Several systems are available for endoscopically grading the extent of injury to the GI tract following corrosive ingestion. The most commonly used system is the Zargar grading system[4].

Figure 4 – corrosive injury to the esophagus showing sloughing of squamous epithelium in a linear pattern along with edema of the mucosa

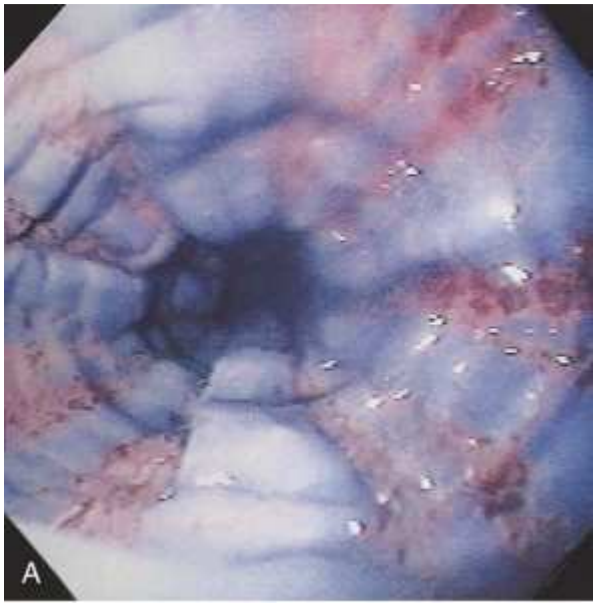


Figure 5 – corrosive injury to the stomach showing edematous and haemorrhagic mucosa.

Table 5 – Zargar grading system for Corrosive injury to the GI tract

Grade	Visible appearance	Clinical significance
0	History positive, no symptoms and visible damage	Able to take fluids immediately
I	Edema, hyperemia, loss of normal mucosal pattern, no transmucosal injury	Temporary dysphagia, able to swallow liquids in 0 – 2 days, no long term sequelae
II a	Transmural injury, friability, blistering, exudates, haemorrhage, scattered superficial ulceration	Scarring, no stenosis, no long term sequelae
II b	2a plus deep discrete ulceration and/or circumferential ulceration	Small risk of perforation, scarring may result in later stenosis (75%)
III a	Scattered deep ulceration with necrosis of tissue	Risk of perforation, High risk of later stenosis (70 – 100%)
III b	Extensive necrotic tissue	High risk of perforation and death(65%), High risk of stenosis

Other grading systems which can also be used are Kikendall grading system and Hollinger Fridman grading system. Sometimes a grade IV is used to describe a perforation.

Other diagnostic modalities that can be used:

Endoscopic ultrasonography – may be superior to conventional endoscopy in its ability to assess the depth of injury beneath the gastrointestinal mucosa induced by the corrosive which is not reliably assessed by the latter[17].

Computed tomography scanning – there is a role for CT in detecting extraluminal air in the body cavities as a sign of perforation for which it has higher sensitivity when compared to plain or contrast radiographs[33]. CT also has an additional role in visualizing the esophagus and stomach distal to severe corrosive burns for which endoscopy or contrast radiographs may not be safe. Chest CT may also be useful to non-invasively determine the response of strictures to dilation procedures[18].

Sucralfate swallow labeled with Tc99m has been suggested to have a role in assessing esophageal injury after ingestion of corrosive substances[19].

Transabdominal USG was used to evaluate corrosive gastritis in a 10-year-old with acid ingestion. It may supplement other diagnostic modalities in the early management of severely ill patients[20].

More studies are needed to better define the role, if any, of these techniques in assessing caustic injuries.

Predictors of injury

Several studies and analyses have attempted to identify a constellation of signs and symptoms whose presence or absence at presentation correctly predicts the presence of gastrointestinal tract injury and thus the need for endoscopy. Most of these studies have been conducted in the pediatric age group or in young adults and were done in patients with history of alkali ingestion. Studies of the presence or absence of oropharyngeal burns identified on examination as a predictor of distal esophagogastric injury have repeatedly found this finding to be poorly predictive.

A retrospective study of 378 children admitted for a caustic injury found that signs or symptoms could not be used to predict significant esophageal injury. However, one prospective study of 79 children evaluated for vomiting, drooling, and stridor found that a combination of two or more of these signs (Crain score) was a predictor of significant esophageal injury as

visualized on endoscopy, with a 95% confidence interval for the sensitivity of these criteria ranging from 100% down to only 59% [21]. In another retrospective study conducted with 115 children younger than 15 yrs of age revealed the fact that all 20 patients who had complications such as strictures had atleast one sign or symptom in the Crain score supplemented by oral burns and dyspnoea(95% confidence limits for sensitivity ranging from 100 – 85%). A prospective study of alkali ingestions by both adults and children found 100% specificity for stridor in predicting significant injuries in the esophagus, but was based on only three patients with this sign[22].

The abdominal examination is likewise an unreliable indicator of the severity of injury. The presence of abdominal pain suggests tissue injury, but the absence of pain or findings on abdominal examination do not preclude life-threatening gastrointestinal damage. For the prediction of long term complications, especially stricture formation, one study suggested that involvement of the esophagus in its entirety, the presence of hematemesis and increased serum LDH, are useful indicators for the occurence of strictures[25].

Management

Acute management:

Basic decontamination and observation of universal precautions by the examiner essentially form the first step in patients with history of corrosive ingestion. Airway inspection and protection should be followed by basic resuscitation principles.

Immediate assessment and stabilization of the airway are essential in patients with stridor, dyspnea, or evidence of significant oropharyngeal swelling. Health care providers should be protected from secondary exposure by personal protection equipment such as gowns, gloves, and goggles. In patients with signs of life-threatening upper airway involvement, direct visual evaluation of the pharynx and larynx with a direct or a fibreoptic laryngoscopy, with sedation as necessary, should be undertaken immediately as blind nasotracheal intubation attempts always carry the risk of perforation of edematous tissues of the pharynx and larynx. An endotracheal tube (generally 1 to 2 mm smaller than usual) should be placed if significant laryngeal edema is identified.

Both tracheostomy and cricothyrotomy can interfere with the surgical field if esophageal repair is required following corrosive ingestion. Hence non-

surgical airway management is usually recommended whenever possible[24]. However in case of patients with severe airway obstruction requiring surgical management, the decision depends upon the clinical status of the patient, the ability to successfully secure an airway via a laryngoscope and the comfort of the physician performing the procedure. In patients with signs of corrosive-induced airway edema steroids in the dose of dexamethasone 10 mg IV in adults and 0.6 mg/kg up to a total dose of 10 mg in children is widely used though studies are not available. A proactive, anticipatory approach is warranted, an approach paralleling airway management in cases of infectious epiglottitis or thermal airway burn.

Similarly, aggressive volume resuscitation and circulatory support may be needed as both acid and alkali ingestions can cause third spacing of intravascular fluid into the interstitial space resulting in hypotension. Individual fluid requirements should be based upon the clinical assessment of neck vein distension and central venous pressure measurement. Patients should be kept NPO and given analgesia as needed[32].

Decontamination, dilution and neutralization:

Removal of any residual corrosive agent by copious and careful irrigation of the patient's skin and eyes forms the essential step in decontamination of the patient. Gastrointestinal decontamination in the form of induced

emesis (as it carries the risk of reintroducing the corrosive to the upper gastrointestinal tract and airway) or activated charcoal (as most corrosives are not adsorbed to activated charcoal and it also interferes with endoscopic tissue evaluation and precludes a subsequent treatment plan) is contraindicated.

Gastric emptying via a narrow, carefully placed nasogastric tube may be attempted with gentle suction in case of patients with history of acid consumption who present within 30 minutes. Although this technique has carries the risk of perforation, it can be tried for removing residual acid from the stomach in patients with intentional ingestions of large amount of acid as these patients have a grave outcome with limited treatment options. Gastric emptying in these cases prevents absorption of some portion of the ingested acid and hence has potential benefit in reducing systemic toxicity and hence has a favorable risk-to-benefit analysis in case of presumed lethal acid ingestions.

In contrast, in case of alkaline and unknown caustic ingestions there is a definite risk of perforation that clearly outweighs the benefit for a nasogastric tube to be blindly passed.

Models assessing the efficacy of dilutional therapy in corrosive ingestions it have suggested the utility of milk or water administered within few

seconds to minutes following ingestion to attenuate the histological damages. This is explained theoretically by the fact that dilutional therapy minimizes gastrointestinal mucosal damage by decreasing the concentration of acid and washing solid adherent preparations away from the gastrointestinal mucosal surface. However, damage to the mucosa from the acid is believed to occur immediately, and thus the utility of dilutional therapy is questionable. Delayed dilutional therapy may be of value for solid substances (eg, crystal lye) rather than liquid substances, as solid agents have increased contact time with the tissues and they usually have a relatively higher concentration over a small surface area. In such cases an optimal agent to be used for attenuating the heat generated by a corrosive may be milk.

Dilutional therapy should generally be limited to patients who present within the first few minutes after ingestion, with no evidence of airway compromise, who are otherwise alert and asymptomatic. In patients with alarming signs such as nausea, stridor, drooling, or abdominal distension dilutional therapy is best avoided as in such cases it may stimulate vomiting and cause the corrosive being reintroduced to the upper gastrointestinal tract. Finally, dilutional therapy has not been appropriately evaluated in human studies that cannot be conducted for ethical reasons.

Neutralization of corrosives should likewise be avoided as this technique can generate an exothermic reaction and worsen the tissue damage or result in the production of gas which can distend and further disrupt an already damaged esophagus.

Surgical management:

The presence of perforation as evidenced clinically by severe abdominal rigidity/ileus or perforation or proven by endoscopic or radiological investigations makes the decision to perform surgery in patients with corrosive ingestions obvious. Hypotension if present is a sign of perforation or significant blood loss and is a grave finding. Several patients despite having impending perforation, necrosis, sepsis, or delayed hemorrhage will not have an obvious indication for surgical intervention.

Several studies have been conducted to identify the constellation of signs which when present can identify patients rapidly in whom surgical treatment may be beneficial in the absence of clear clinical indications. These studies have put forth that patients with history of ingestion of large amounts (>150 mL) and those patients presenting with shock, acidemia, or coagulation disorders had severe findings on surgical exploration[24]. Some surgeons also advocate surgical exploration for patients with third degree burns on endoscopy[27]. Zargar *et* al have suggested that in patients with

grade 3b injuries mortality and morbidity may be improved by prompt surgical resection. The poor predictive value of abdominal examination in identifying those with the need for surgery was also reinforced by these studies.

Surgical intervention in the form of laparotomy or thoracotomy may be required for tissue visualization, resection, and repair of perforations. In the presence of perforation emergency surgery in the form of esophagectomy or gastrectomy is needed with colonic interposition being additionally required sometimes. Laparoscopy may also be used, but inspection of the posterior aspect of the stomach may not be possible.

Controversies exist regarding the necessity and timing of operative intervention in patients with severe ulceration or necrosis in the absence of definitive evidence of perforation. Suggestions are present for lower mortality with both early operative management as well as non-operative supportive. Hence surgical management in these patients must be considered on an individualized basis.

Sub acute managmement

Once the patients have been stabilized and perforation has been ruled out by taking chest and abdominal radiographs, they should be subjected to

Upper GI endoscopy for grading the extent of injury which will further decide the plan of management.

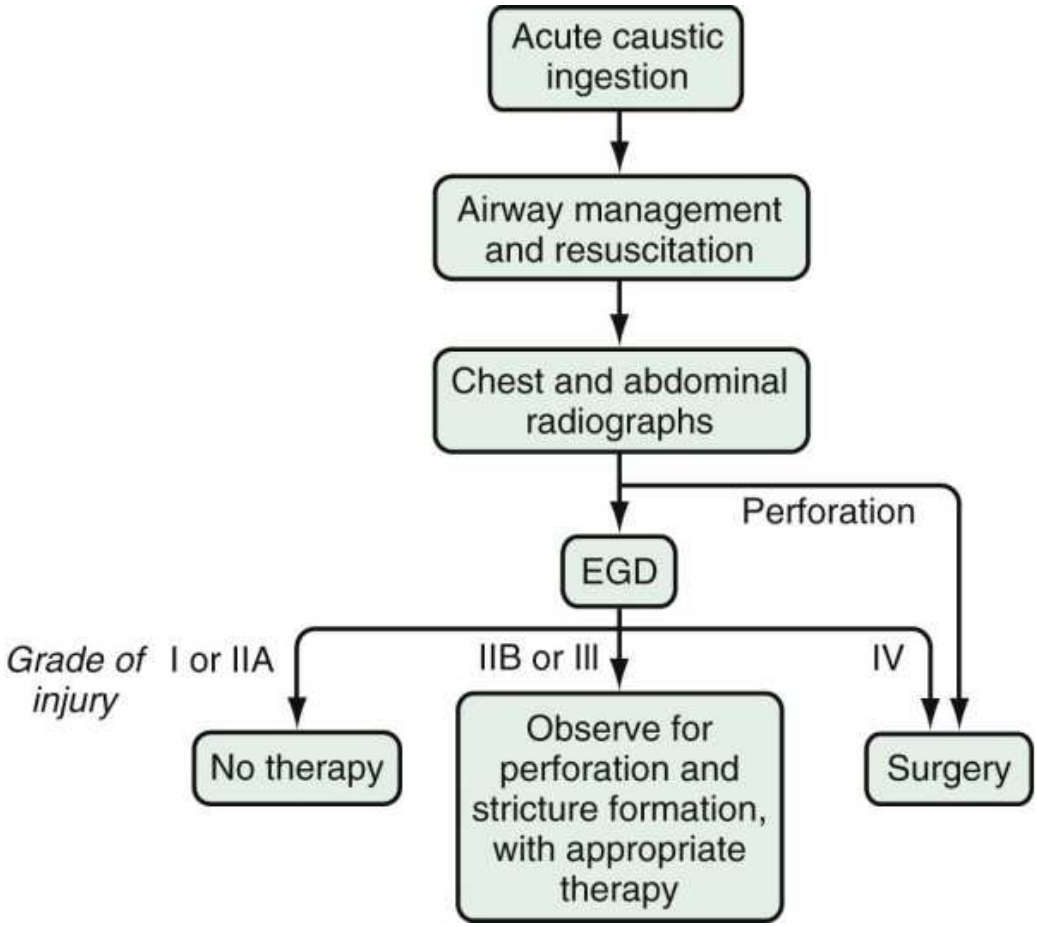
Asymptomatic patients with normal endoscopic examination or isolated grade I injuries have no risk of delayed complications such as stricture or carcinoma. Their diet can be resumed as tolerated in the first 24 – 48 hours. These patients can be discharged once they are able to eat and drink normally and are psychiatrically stable[32].

Patients with grade II a and some patients with grade II b injuries recover rapidly and may be discharged from the hospital within 5 – 12 days. Healing usually occurs by 3rd or 4th week without any sequelae. Nutrition in such patients can be achieved by nasogastric intubation preferably under direct endoscopic visualization or under fluoroscopic guidance. Nasogastric tube placement in such patients has certain distinct advantages such as

- providing adequate nutritional support and medications
- protecting the esophagus by giving rest and preventing wound trauma be associated with bolus food ingestion and
- maintaining a lumen that can be used to assist dilation if at all strictures should occur in patients with higher grades of injury.

Regular follow-up with endoscopy or with X rays for the complications of perforation, infection, and stricture development should be done for patients with grades IIb and III lesions. Patients with grade III burns, in particular, have the risk of progression to stricture formation regardless of therapy along with other complications such as infection, fistula formation, and perforation with associated mediastinitis and peritonitis.

Figure 6 - A step wise approach to a patient with history of corrosive ingestion[32]



Strictures occur due to scar formation in the injured tissues through the production of collagen. Strictures are a debilitating complication of corrosive ingestions usually occurring over a period of weeks or months. Several treatment strategies have been suggested to prevent stricture formation and consequent esophageal obstruction. These include the use of corticosteroids, antibiotics or the placement of esophageal stents or nasogastric tubes.

Role for steroids:

The role of steroids in preventing stricture in patients with grade II b and grade III continues to be a controversial issue. It has been hypothetised that steroid therapy arrests the process of inflammatory repair and hence can potentially prevent stricture formation. Corticosteroids affect the ability of a wound (i.e., burn) to heal by decreasing the amount of collagen in the tissue and inhibiting the inflammatory process initiated early after the burn injury. Cellular collagen content is diminished through the inhibition of enzymes affecting synthesis (i.e., prolyl hydroxylase) and crosslinking (i.e., lysyl oxidase), as well as stimulation of degradative activity through collagenase activation. Corticosteroids also exert their effect on the inflammatory process by diminishing the amount of eicosanoids synthesized through inhibition of the arachidonic acid

formation taking place within the membranes of the cells. Based on these mechanisms, their use in the prevention of stricture formation after acid ingestion seems rational.

In studies evaluating the efficacy of steroid use for the prevention of stricture, the results were not supportive for the use of steroids. They noted an increase in the adverse effects of steroids such as increased susceptibility to infection and increased risk of GI bleeding[26]. Most studies find that first-degree burns of the esophagus do not progress to strictures whether treated with corticosteroids or not, whereas third-degree burns do progress to stricture formation regardless of corticosteroid therapy. In a prospective, randomized, controlled trial conducted in children with history of corrosive ingestion showed that corticosteroids were not effective in decreasing the occurrence of strictures though the need for surgery (total esophagectomy) was lesser in the group treated with steroids (four vs. seven untreated patients).

Methyl prednisolone is the agent of choice at a dose of 40 to 60 mg/ day intravenously given usually for duration of at least 3 weeks and then tapered off over a period of 4 – 6 weeks. Ampicillin is usually added to mitigate secondary inflammation due to bacterial invasion of injured tissue.

Role of antibiotics:

The use of antibiotics alone for preventing strictures has not been majorly investigated. It is generally agreed to use an antibiotic (usually Ampicillin) in patients who are treated with steroids. The prophylactic use of antibiotic, in the absence of steroid therapy, is not advocated.

Esophageal stents and nasogastric tubes

Intraluminal silicone rubber stents or nasogastric tubes can be used for maintaining the esophageal luminal patency. They can be retained for a period of 3 weeks following which the esophageal lumen can be reassessed for the presence of strictures. However esophageal stents are not without potential disadvantages, which interfere with healing, in the form of mechanical trauma at the site and increased reflux. Nasogastric tubes likewise can be placed early in the course of treatment for ensuring the patency of the lumen. But NG tubes also carry the risk of increasing fibrosis and stricture formation by causing irritation and increasing inflammation of the healing esophagus. Nevertheless an NG tube can be placed as it allows further endoscopic dilatation and avoids trauma from food bolus.

Proton pump inhibitors (PPIs) and H2-blockers:

Parenteral PPIs and H2 blockers also have a role in limiting ongoing injury to the injured GI mucosa. Hence they are being routinely recommended in corrosive ingestion[31].

Additionally, several xenobiotics have been studied in various animal models which can prevent stricture formation by interfering with collagen synthesis or enhancing its destruction. Some examples are β-Amino propionitrile (BAPN), heparin, halofuginone, N-acetylcysteine (NAC), vitamin E, epidermal growth factor (EGF), penicillamine, caffeic acid phenethyl ester (CAPE) and colchicine. There have also been reports advocating the effects of other agents like sucralfate and the use of TPN (Total Parenteral Nutrition) in decreasing the incidence of stricture formation but more studies are needed to delineate their beneficial action. Early dilatation is associated with increased risk of perforation and is hence discouraged. The presence of co existent GERD accelerates the formation of stricture by worsening the corrosive insult to the esophagus and hence periodic screening for GERD is recommended in patients with corrosive ingestion.

Management of long term complications

Esophageal stricture can develop in as much as one third of corrosive ingestion patients after initial recovery. The most common period for stricture formation to present is at two months after injury but it can occur over a wide period ranging from two weeks to as many as years after the initial injury. Stricture formation is more common following more severe (grade IIB or III) injuries. Commonly, the management of esophageal strictures includes early endoscopic dilation using various types of dilators[32].

Maximal esophageal wall thickness, as determined by contrast CT is one parameter which has several applications such as determining long-term follow-up, type of nutritional support, predicting the response to dilatation and the number of sessions required to achieve adequate dilation, in identifying those patients in whom dilations should be done under fluoroscopy to limit the risk of perforation and finally in assessing the potential need for surgical repair as an alternative to dilations. Multiple dilations may often be necessary.

The initial esophageal dilation is best delayed for at least 4 weeks post-ingestion, to allow for the esophagus to heal, and allow remodelling, and potential stricture formation in the esophagus to have taken place, thereby

reducing the risk of perforation associated with dilatation. When perforation occurs during dilatation, patients may complain of sudden onset of shortness of breath or chest pain and show signs of subcutaneous emphysema or pneumomediastinum. Diagnostic imaging may be needed to identify the perforation and provide information for emergent surgical repair if the diagnosis is unclear. Long-term endoscopic follow-up is required in patients with stricture formation for assessing the occurence of neoplastic changes of the esophagus that may occur with a delay of several decades.

Antral and pyloric strictures may also occur after corrosive injury usually at one to six weeks after ingestion, but can also occur years later with the risk of being directly related to the degree of injury. Antral strictures can be successfully managed in many patients with endoscopic dilation and acid suppression. However surgical treatment in the form of antrectomy may be required in some patients[32].

METHODOLOGY

MATERIALS AND METHODS

SUBJECTS

The study was conducted on 50 cases admitted at the toxicology ward in Rajiv Gandhi Government General Hospital, Chennai, with alleged history of consumption of corrosive ingestion.

PERIOD OF STUDY

6 months

DESIGN OF STUDY

Cross-sectional study

CONSENT

Informed consent from all the patients

METHOD OF COLLECTION OF DATA

Inclusion criteria

- Patients age > 12 yrs
- Patients with history of corrosive ingestion presenting within 24 hours of ingestion
- Upper GI endoscopy done in patients within 24 hours of admission

Exclusion criteria

- Patients presenting after 24 hours of corrosive ingestion
- Patients with respiratory distress
- Patients with suspected perforation either radiologically or endoscopically (grade III b injury)
- Patients with normal findings in Upper GI endoscopy (no evidence of initial injury)

METHODOLOGY

All patients who were admitted with history of corrosive ingestion underwent thorough history taking and detailed clinical examination after initial stabilisation of Airway, Breathing and Circulation. The parameters taken into consideration were history regarding amount consumed, type of corrosive, and duration since consumption, symptomatology, physical signs, upper GI endoscopy findings and they were correlated with outcome. Laboratory investigations including Complete blood counts, renal and liver function tests were done in all patients. Chest and Abdomen X rays were taken to rule out perforation. Patients were kept Nil per oral and subjected to Upper GI endoscopy within 24 hours of admission. The findings were noted and patients were managed accordingly (oral feeds within 24 hours for normal and grade I – II a injuries and NG tube

placement for grade II b – III a injuries and feeding jejunostomy for duodenal injuries).

The patients were serially followed and were subjected for a re-look upper GI endoscopy after 6 weeks and the findings were compared and the outcome was graded into 2 categories.

Category I – normal endoscopy study

Category II – stricture esophagus or stricture antri or pylori.

INVESTIGATION DETAILS

Complete history and thorough physical examination in all patients with history of corrosive ingestion and routine blood investigations, chest and abdomen X rays and Upper GI endoscopy.

DATA COLLECTION AND METHODS

Collection of data as per proforma with consent from patients with history of corrosive ingestion in Toxicology ward, Rajiv Gandhi Govt. General hospital.

ANALYSIS

Data analysed using statistical package-SPSS software

Conflict of interest: Nil.

Financial data: Nil

Table 6

GRADING OF INJURY AS PER ZARGAR GRADING SYSTEM:

Grade 0	Normal endoscopy findings
Grade I	Mucosal edema and hyperaemia
Grade II a	Friability, blisters, hemorrhages, erosions, whitish membranes, exudates, superficial ulcerations
Grade II b	Grade 2a plus deep discrete or circumferential ulceration
Grade III a	Small scattered areas of multiple ulcerations and areas of necrosis (brown-black or grayish discoloration)
Grade III b	Extensive necrosis

STATISTICAL ANALYSIS

Statistical analysis was carried out for 50 patients with history of corrosive ingestion after categorizing each variable – age sex, type of corrosive consumed, duration since consumption, amount consumed, circumstance of consumption, symptomatology, presence of physical signs and upper GI endoscopy findings. Datas were analysed using Statistical package- SPSS software version 11.5.The values are presented as mean, standard error of mean, standard deviation, standard error of mean and median. Percentages were used to describe the proportions of discrete variables. The significance of difference between the proportions was indicated by the Chi square (x2) statistic. The significance of difference in mean between the groups was calculated by Fisher exact test. Variables were considered to be significant if $P<0.05$.

OBSERVATION AND RESULTS

OBSERVATIONS AND RESULTS

50 cases with history of corrosive ingestion and with positive findings on endoscopy formed the study group. In these patients age wise distribution, sex wise distribution, circumstances of poisoning (suicidal/accidental), agent of exposure (acid/alkali), symptomatology, physical findings and endoscopy findings were analysed.

The upper GI endoscopy findings were compared with the final outcome. The agent exposed to and the circumstances of poisoning were compared with the final outcome. Other independent variables were entered into the comparison model and appropriate statistical was made.

AGE AND SEX DISTRIBUTION

TABLE 7:

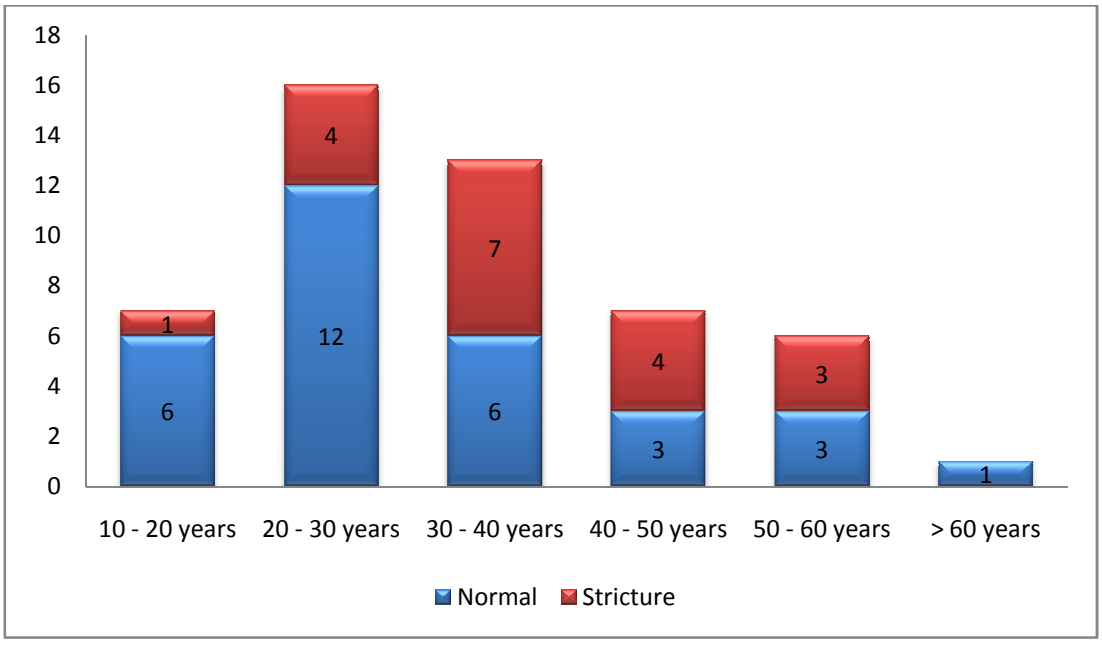
Age in years	Total	%	Male (%)	Female (%)
10-20	7	14	2(5.5)	5 (35.7)
20-30	16	32	13(36.1)	3 (21.4)
30-40	13	26	11(30.5)	2(14.2)
40-50	7	14	5(13.8)	2(14.2)
50-60	6	12	4(11.1)	2(14.2)
> 60	1	2	1(2.7)	-
Total	50	100%	36(72)	14(28)

The mean age among the patients was 32.88 ±12.74. The youngest age was 15 years and the oldest was 67 years. There was no significant difference in the outcome among different age groups (p value > 0.05)

TABLE 8: Age wise analysis of outcome

Age in years	n	I	II
10-20	7	6	1
20-30	16	12	4
30-40	13	6	7
40-50	7	3	4
50-60	6	3	3
> 60	1	1	-

Figure 7 – Graph showing age wise distribution



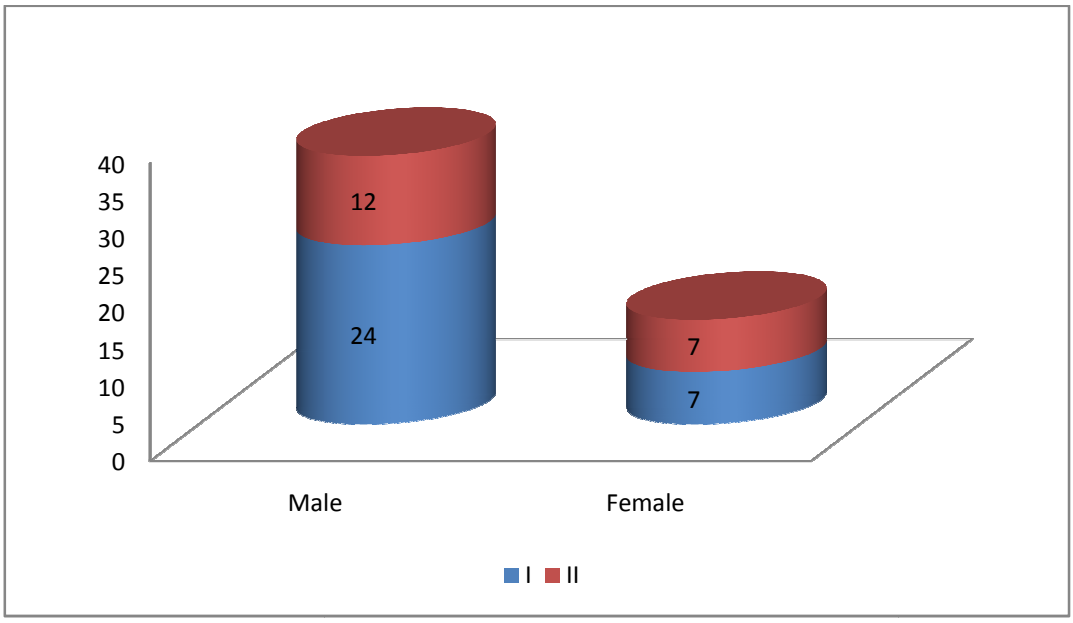
SEX WISE ANALYSIS

Table 9

Sex distribution with outcome

Sex	n	I (%)	II (%)
Male	36	24 (66.7%)	12 (33.3%)
Female	14	7 (50%)	7 (50%)

Figure 8: Graph showing sex wise distribution

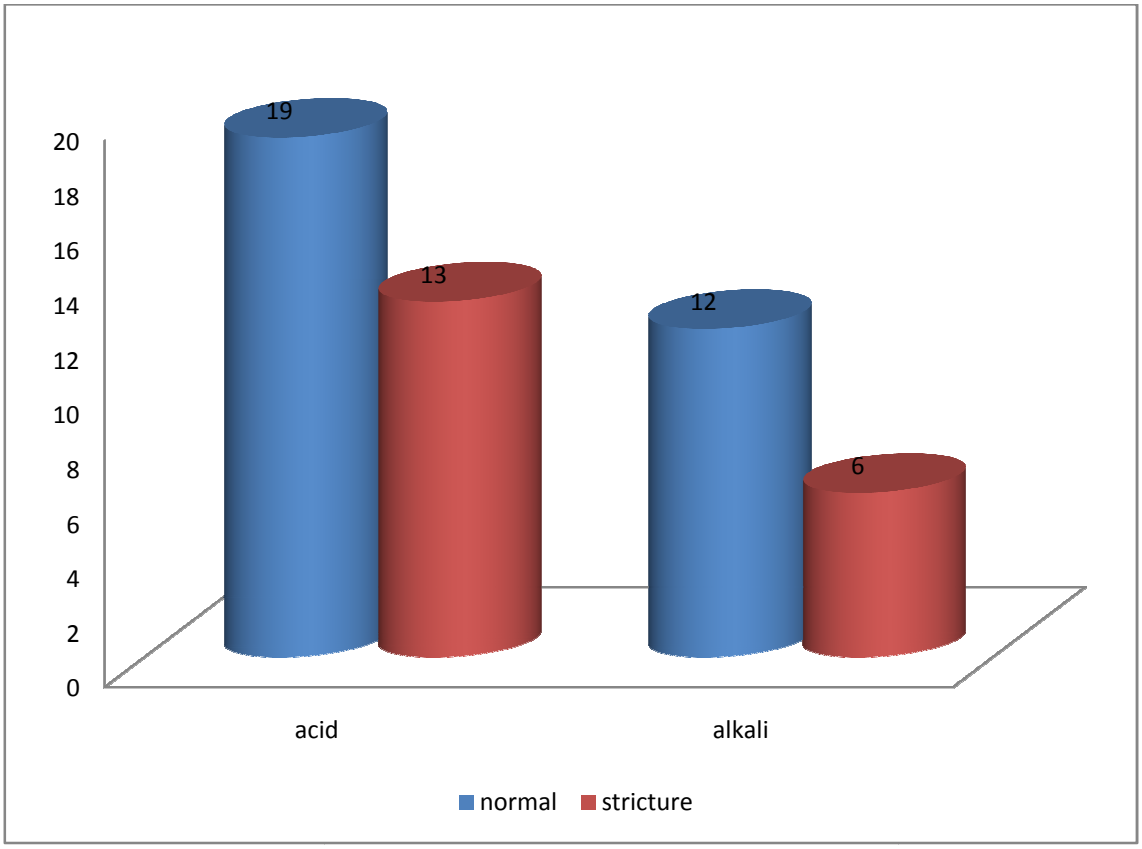


CORROSIVE WISE ANALYSIS

Table 10

Type of corrosive	n (%)	I	II
Acid	32 (64%)	19	13
Alkali	18 (36%)	12	6

Figure 9 – type of corrosive versus outcome



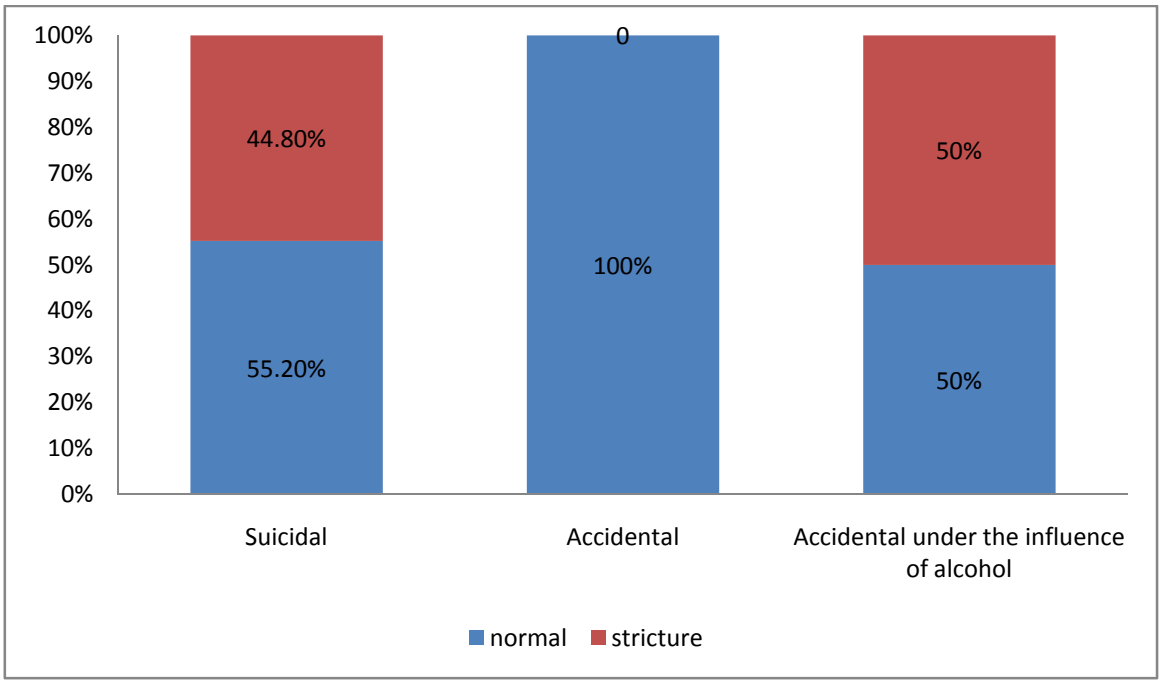
CIRCUMSTANCES OF CONSUMPTION

Table 11 – Frequencies of the circumstances of consumption

Circumstance	n (%)	I	II
Suicidal	38 (76)	21 (55.2%)	17 (44.8%)
Accidental	8 (16)	8(100%)	0
Accidental (under alcohol influence)	4 (8)	2 (50%)	2 (50%)

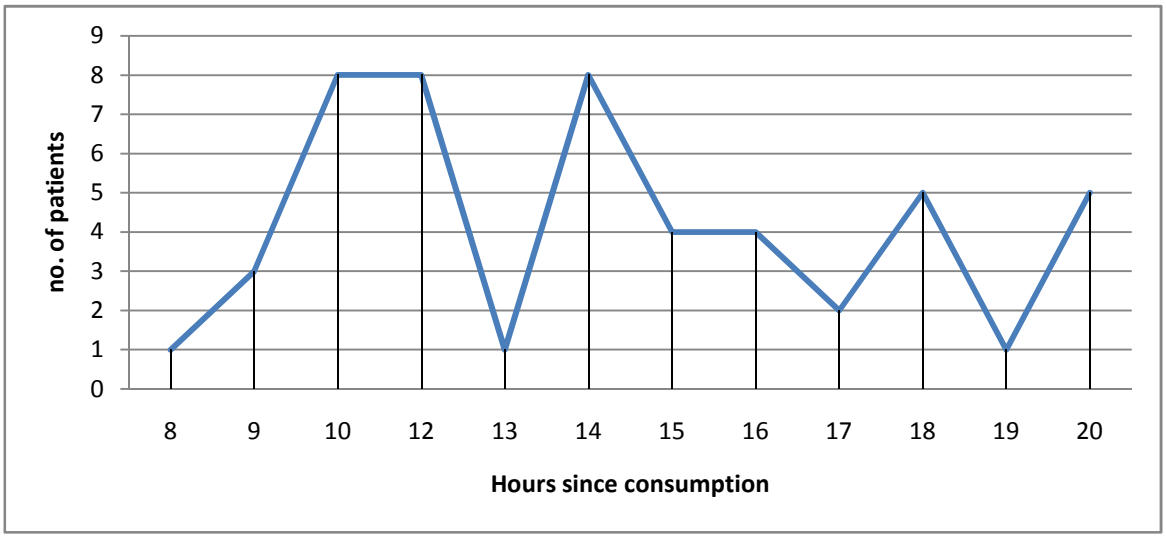
In our study though majority of strictures occurred in the suicidal comsumption group, the difference was not statistically significant (p value – 0.053)

Figure 10 – Circumstance of poisoning versus outcome.



DURATION SINCE CONSUMPTION

Figure 11 – distribution of the duration since consumption when the patient was subjected to upper GI endoscopy.

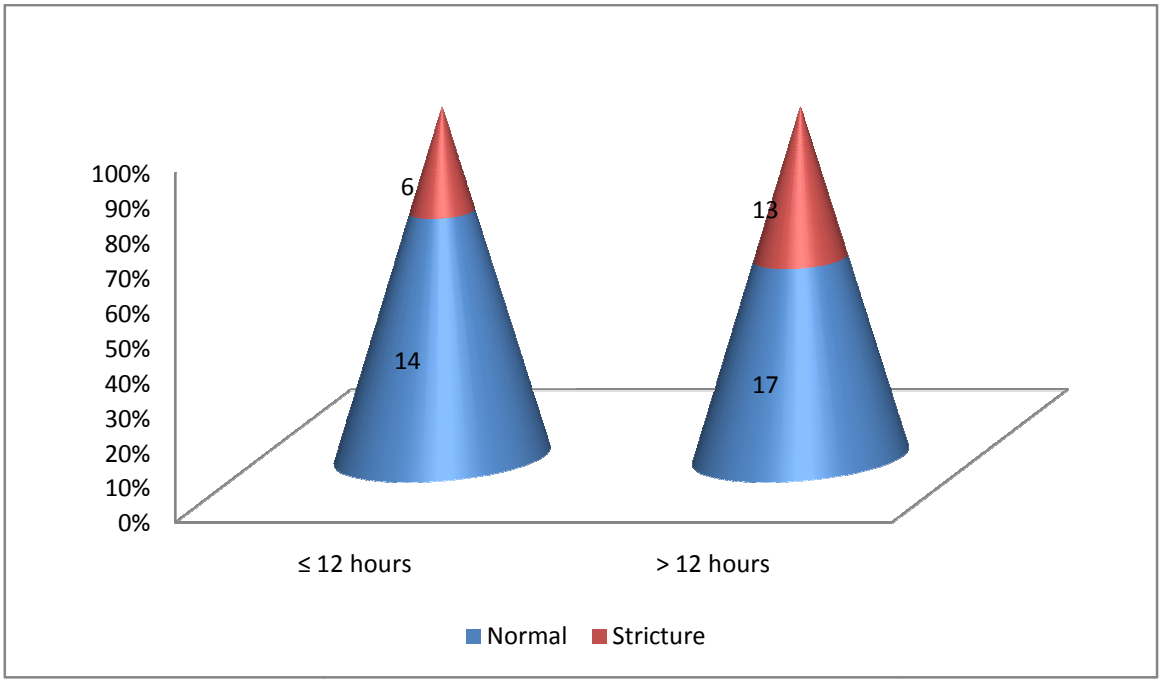


The mean duration for performing the endoscopy was 14.06 ± 3.48 hours. The least duration was 8 hours and the maximum duration was 20 hours. The incidence of strictures was higher in the patients who underwent endoscopy later than 12 hours but the difference was not statistically significant (p value > 0.05).

Table12 – Analysis of duration since consumption versus outcome

Duration since consumption	Frequency	I	II
≤ 12 hours	20	14	6
> 12 hours	30	17	13

Figure 12 – Analysis of duration since consumption versus outcome



AMOUNT OF CORROSIVE CONSUMED WISE ANALYSIS

Figure 13 – distribution of amount of corrosive consumed

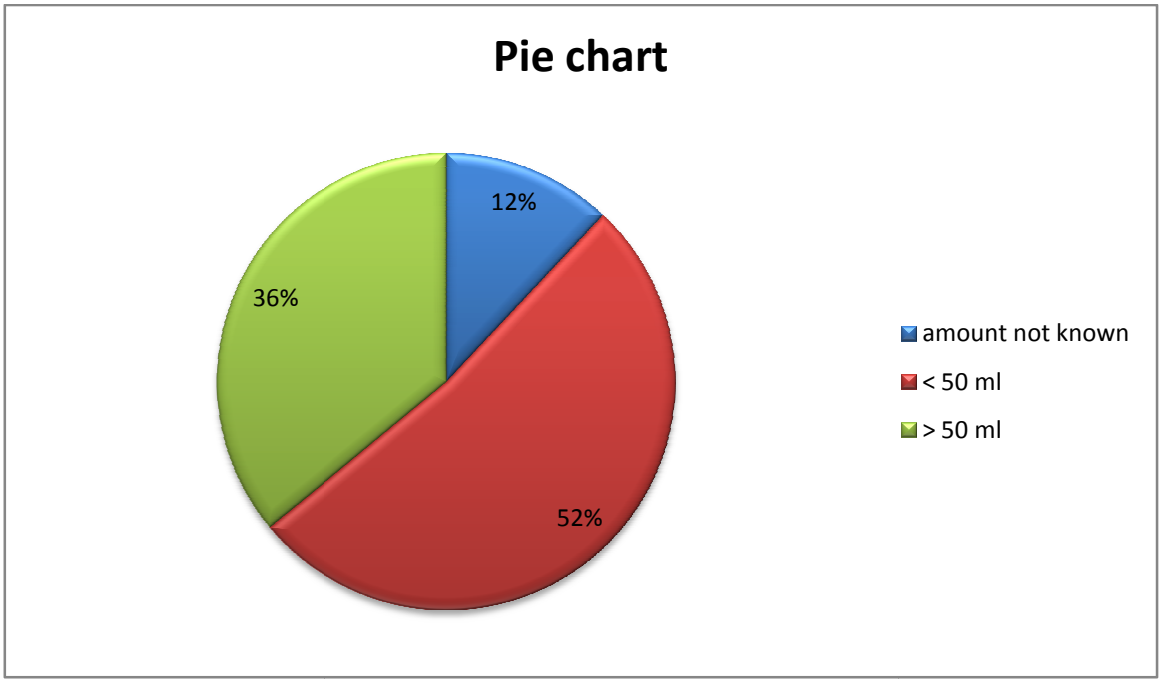
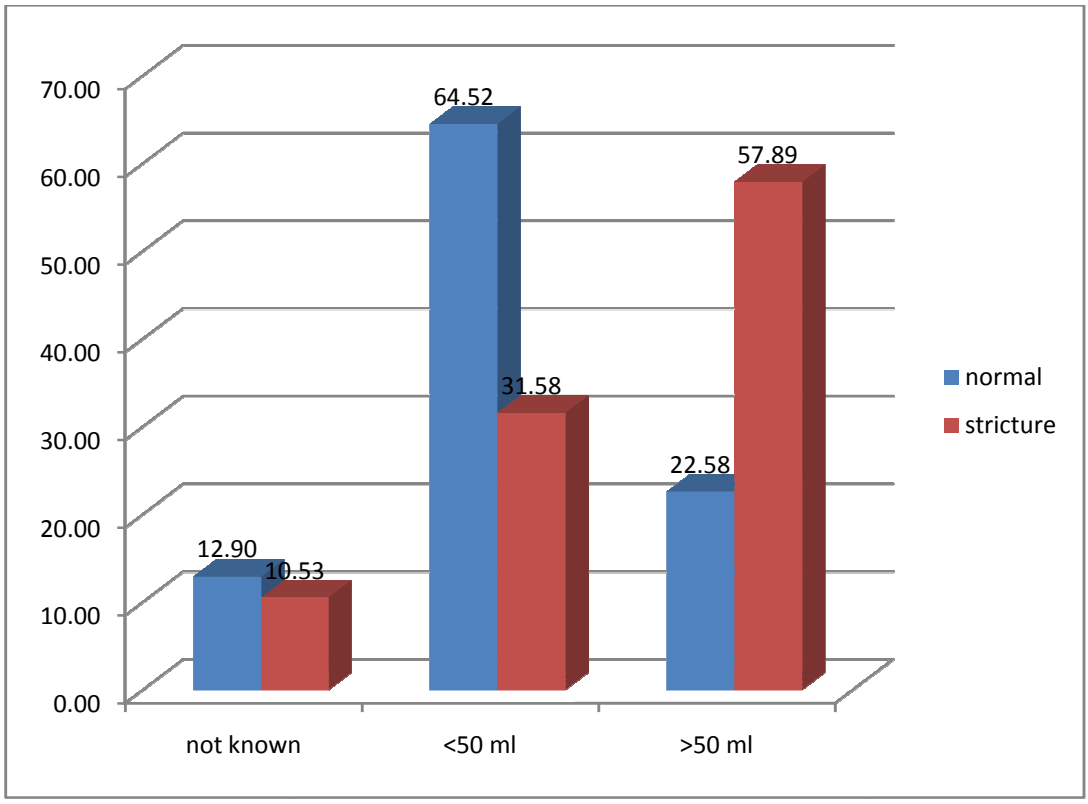


Table 13 - Analysis of outcome based on the amount of corrosive consumed

Amount consumed	n (%)	I (%)	II (%)
Not known	6 (12)	4 (12.9)	2 (10.5)
< 50 ml	26 (52)	20 (64.5)	6 (37.6)
> 50 ml	18 (36)	7 (22.6)	11 (57.9)

Among the 50 patients in our study group, the incidence of strictures was more in the group of patients who had consumed more than 50 ml and the difference was statistically significant (p value – 0.037).

Figure 14 – Graph showing outcome based on the amount of corrosive consumed:



SYMPTOMATOLOGY AT PRESENTATION

Figure 14 – Chart showing distribution of presenting symptoms

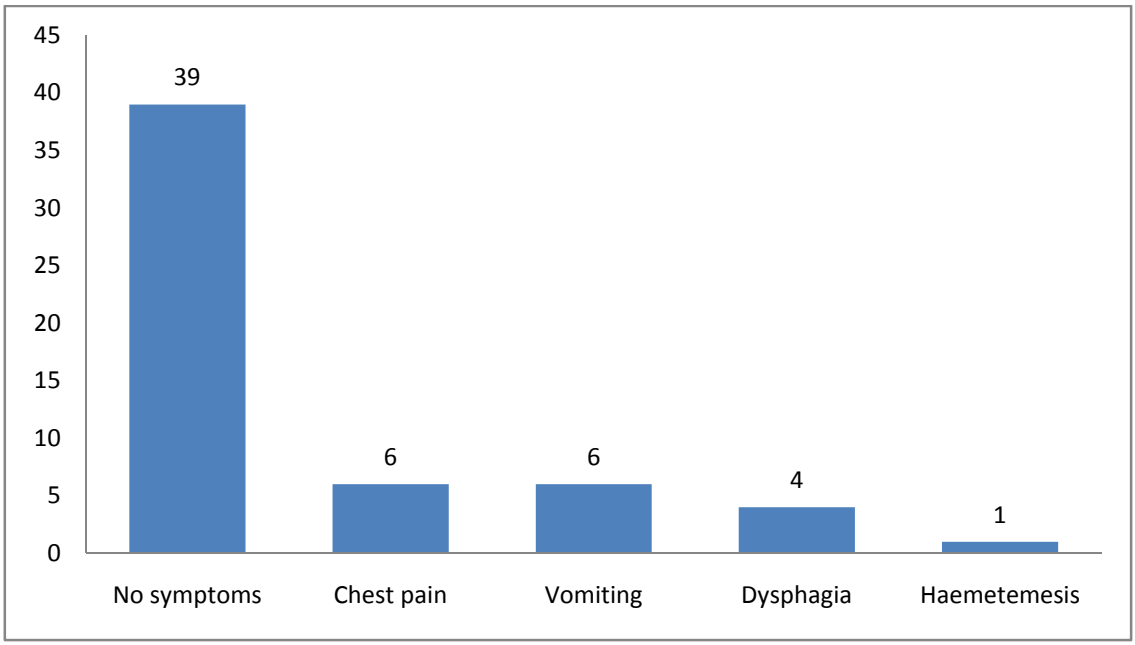


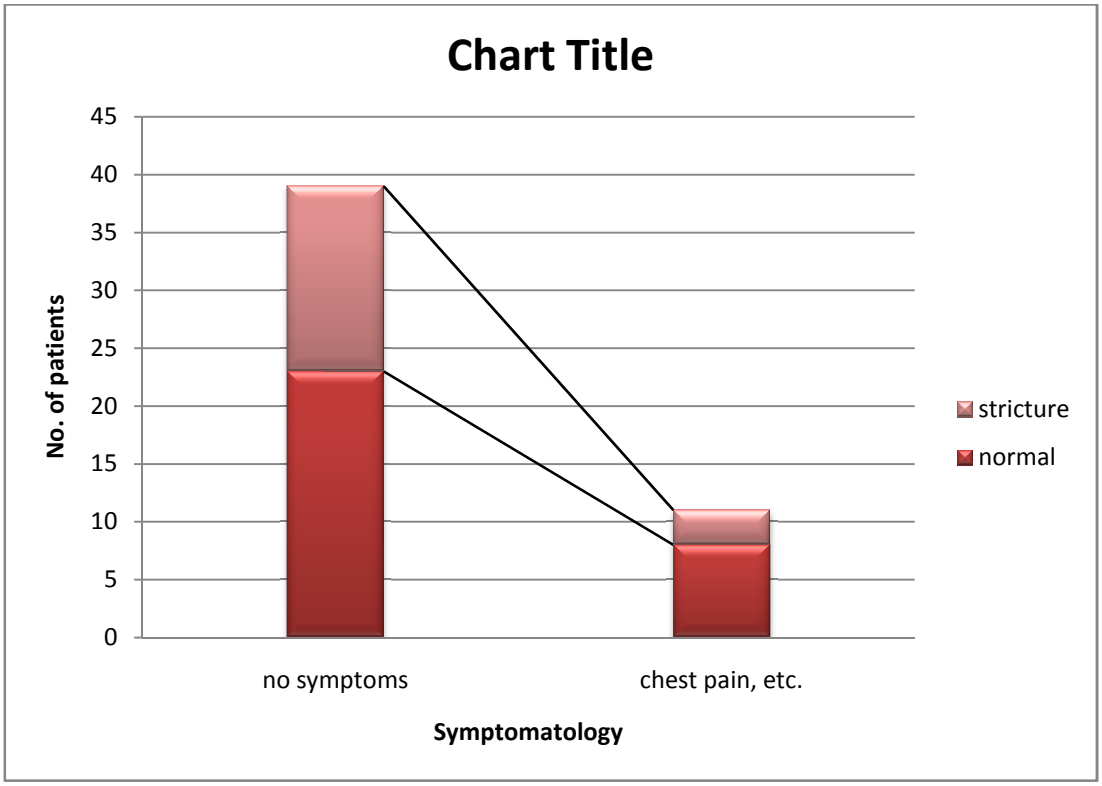
Table 14 – Analysis of outcome versus presenting symptoms

	n (%)	I (%)	II (%)
No symptoms	39 (78)	23 (46)	16 (32)
Chest pain, vomiting, etc.	11 (22)	8 (16)	3 (6)
Total	50	31 (62)	19 (38)

Majority of the patients were asymptomatic. The most common symptoms among the symptomatic group was chest pain and vomiting.

The incidence of strictures was higher in the patients who had no symptoms but the difference was not statistically significant (p value > 0.05).

Figure 15 – Graph showing analysis of symptomatology versus outcome



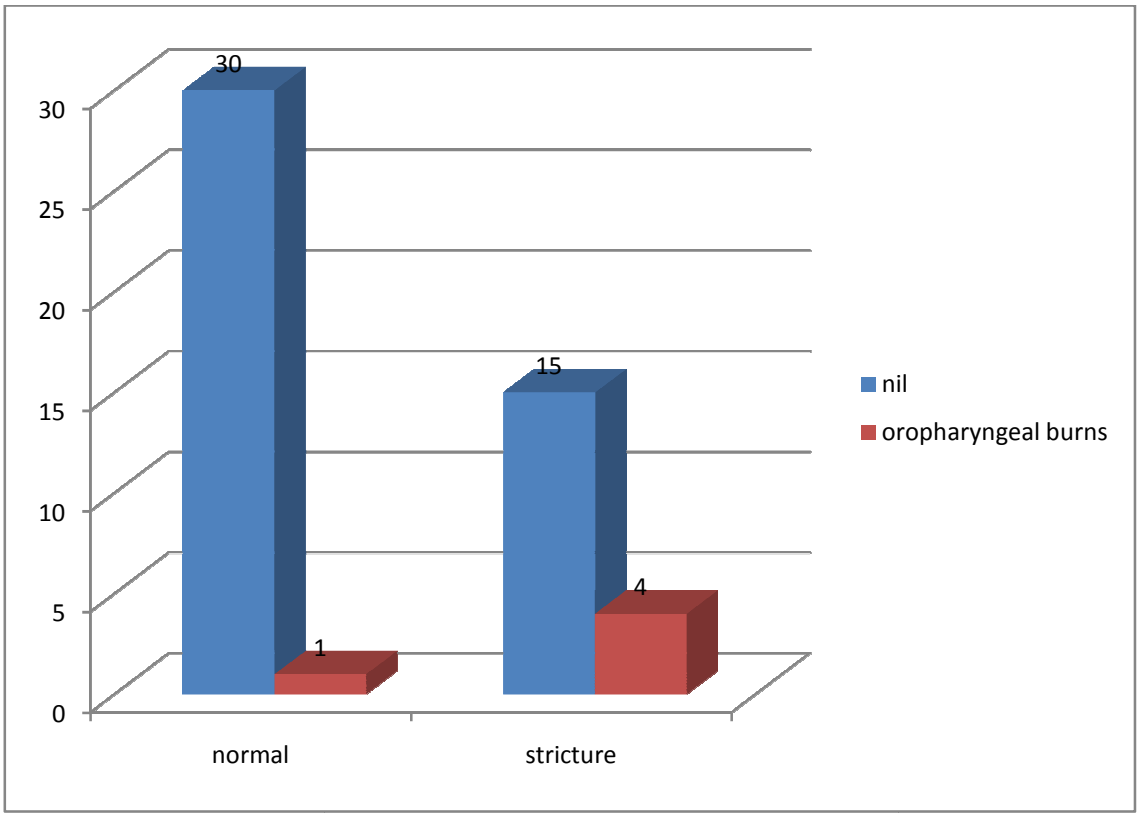
PHYSICAL SIGNS VERSUS OUTCOME

Table – 15 Analysis of physical signs versus outcome

Physical signs	n (%)	I	II
Normal physical examination	45 (90%)	30	15
Oropharyngeal burns	5 (10%)	1	4

The incidence of strictures was higher in the patients with normal physical examination than those with oropharyngeal burns and the difference was statistically significant (p value – 0.043). Hence oropharyngeal burns were not a reliable predictor of injury and long term outcome.

Figure 16 – Graph showing analysis of physical signs versus outcome

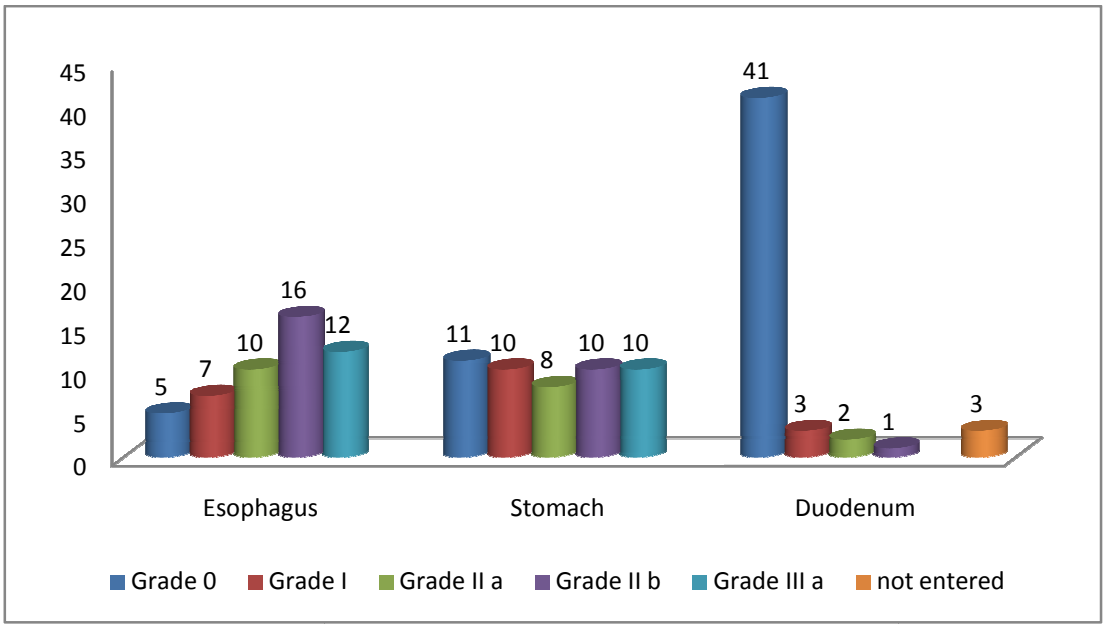


ENDOSCOPY FINDINGS AT ADMISSION

Table 16

Grade of injury	Esophagus	Stomach	Duodenum
Grade 0 (normal)	5	11	41
Grade I	7	10	3
Grade II a	10	8	2
Grade II b	16	11	1
Grade III a	12	10	-
Not entered	-	-	3

Figure 17 – Graph showing distribution of endoscopy findings at admission

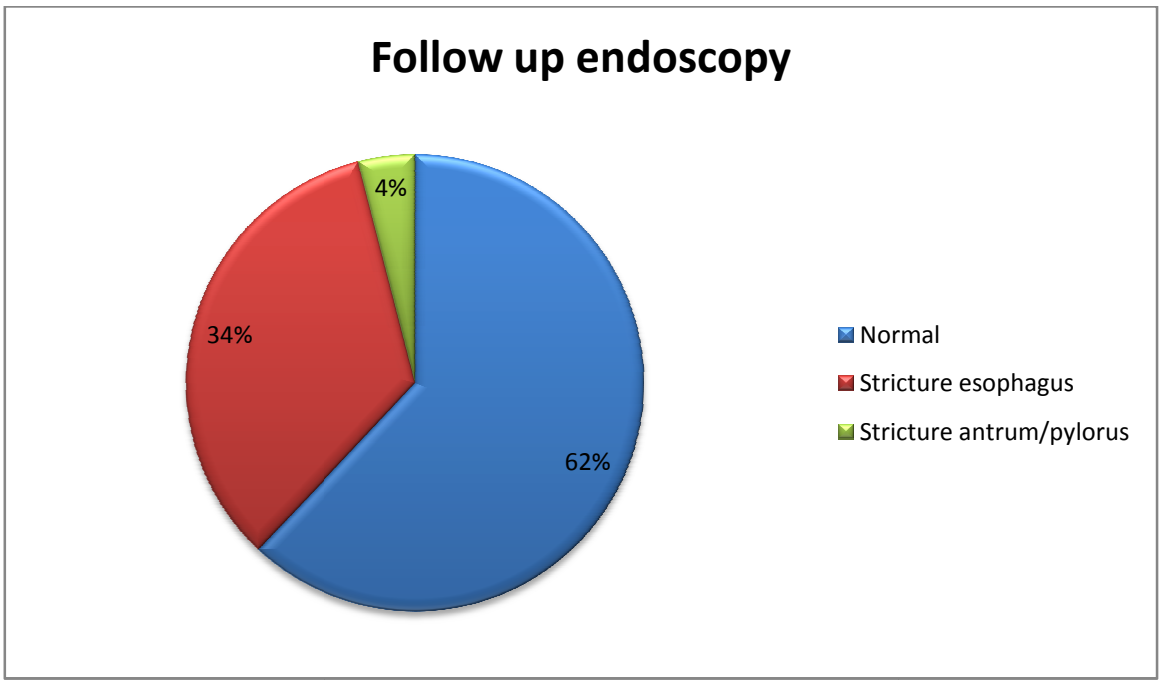


FOLLOW UP ENDOSCOPY

Table 17 – Endoscopy findings at 6 weeks

Endoscopy findings	Frequency	Percentage
Normal	31	62
Stricture esophagus	17	34
Stricture antrum/pylorus	2	4

Figure 18 – Distribution of follow up endoscopy findings



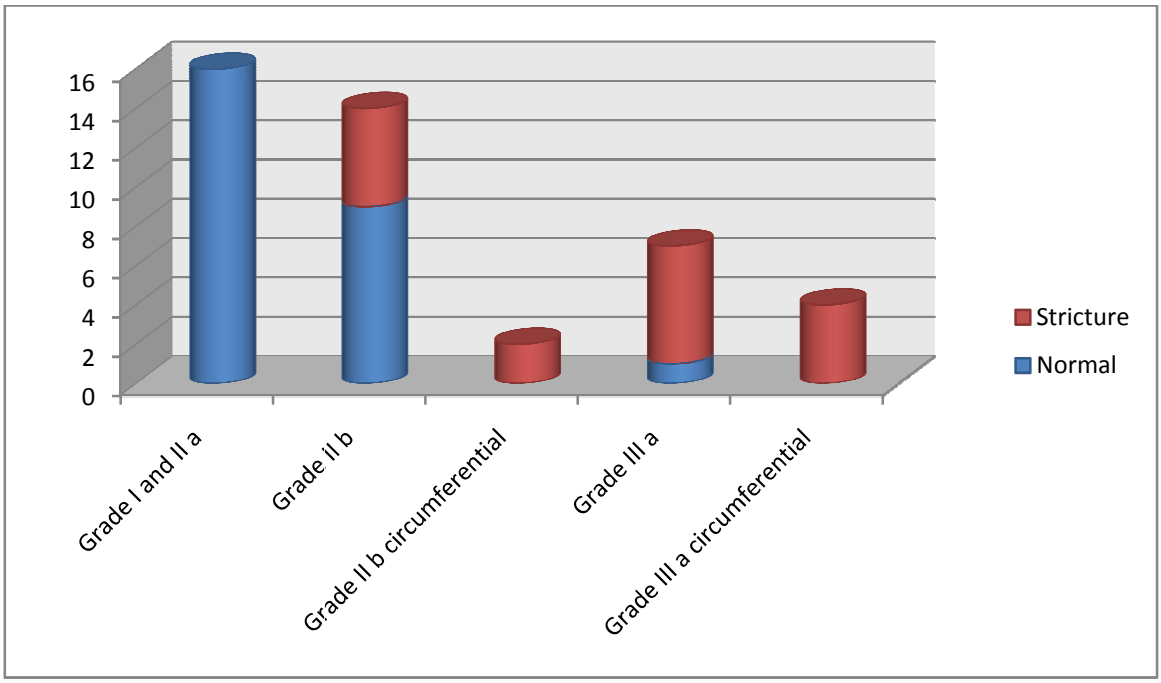
ANALYSIS OF ESOPHAGUS ENDOSCOPY FINDINGS VERSUS OUTCOME

Table18

Endoscopy findings	Frequency	I	II
Grade I	6	6	0
Grade II a	10	10	0
Grade II b	14	9	5
Grade II b circumferential	2	0	2
Grade III a	7	1	6
Grade III a circumferential	4	0	4

The incidence of strictures was higher in patients with Grade II b and III a injuries especially with circumferential injuries and the difference was statistically significant (p value < 0.05). The patients with grade I and II a injuries had no incidence of strictures.

Figure 19 – Analysis of esophagus endoscopy findings versus outcome



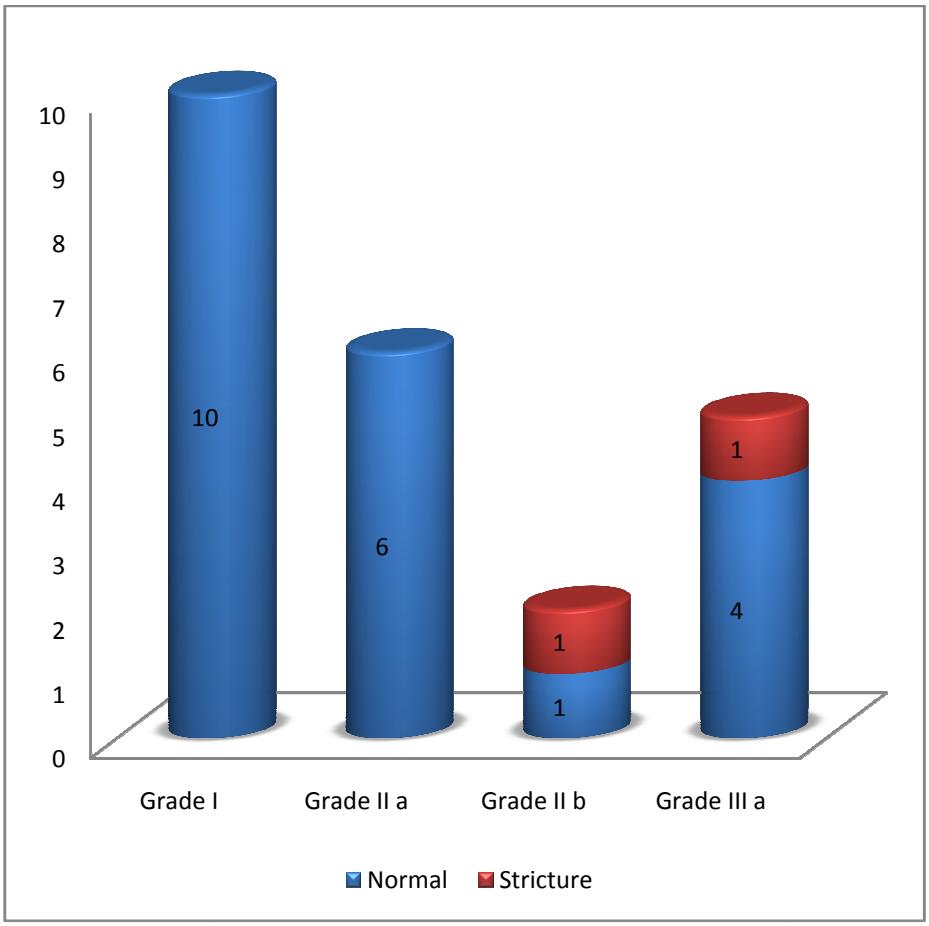
ANALYSIS OF FOLLOW UP ENDOSCOPY FINDINGS IN STOMACH

The 17 patients who had stricture esophagus in the follow up endoscopy were not taken into consideration for assessing the recovery of stomach findings as the endoscope was not passed beyond the strictured part in the esophagus. The remaining patients were analysed for assessing the outcome of stomach injuries. The incidence of stricture was equal in grade II b and III a injuries but the difference was not statistically significant (p value > 0.05)

Table 19 – Analysis of outcome of stomach injuries

Endoscopy findings	Frequency	I	II
Grade I	10	10	O
Grade II a	6	6	0
Grade II b	2	1	1
Grade III a	5	4	1

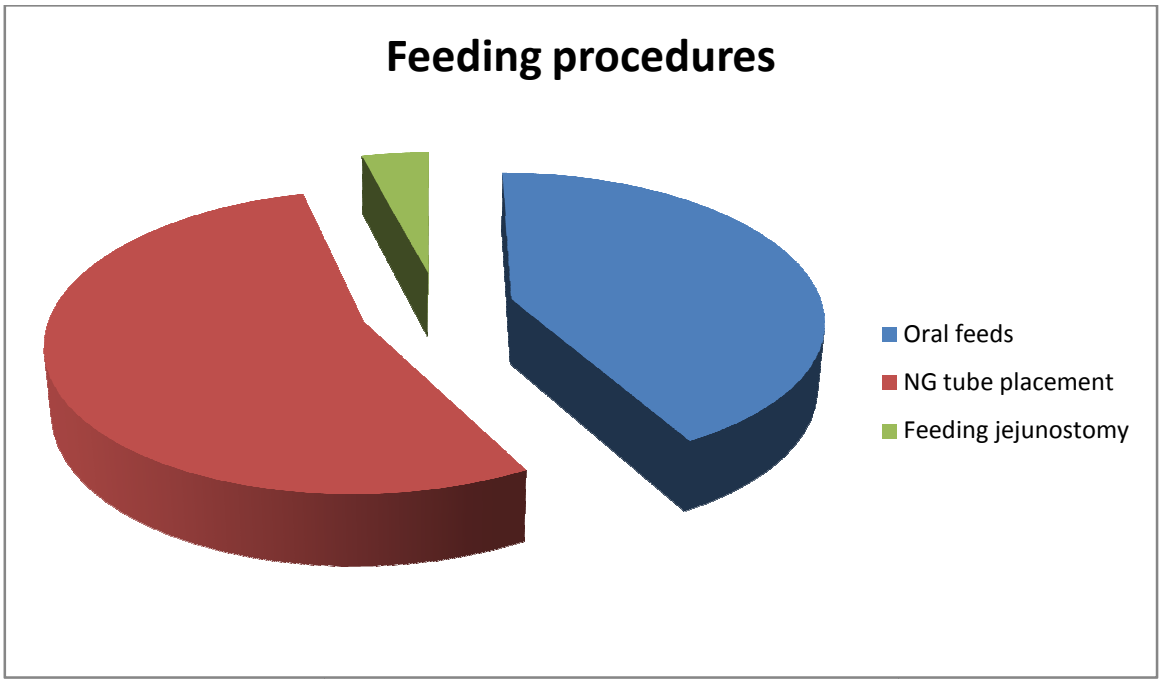
Figure 20 – Outcome of corrosive injuries of stomach



ANALYSIS OF TREATMENT VERSUS OUTCOME

Among the 50 patients in the study group those with grade o, I and II a were started on oral feeds and none of them had stricture in the re-look endoscopy. The patients with grade II b and III a were subjected to nasogastric tube placement under fluoroscopic guidance. Two patients had to undergo feeding jejunostomy.

Figure 21 – Distribution of treatment given at first admission

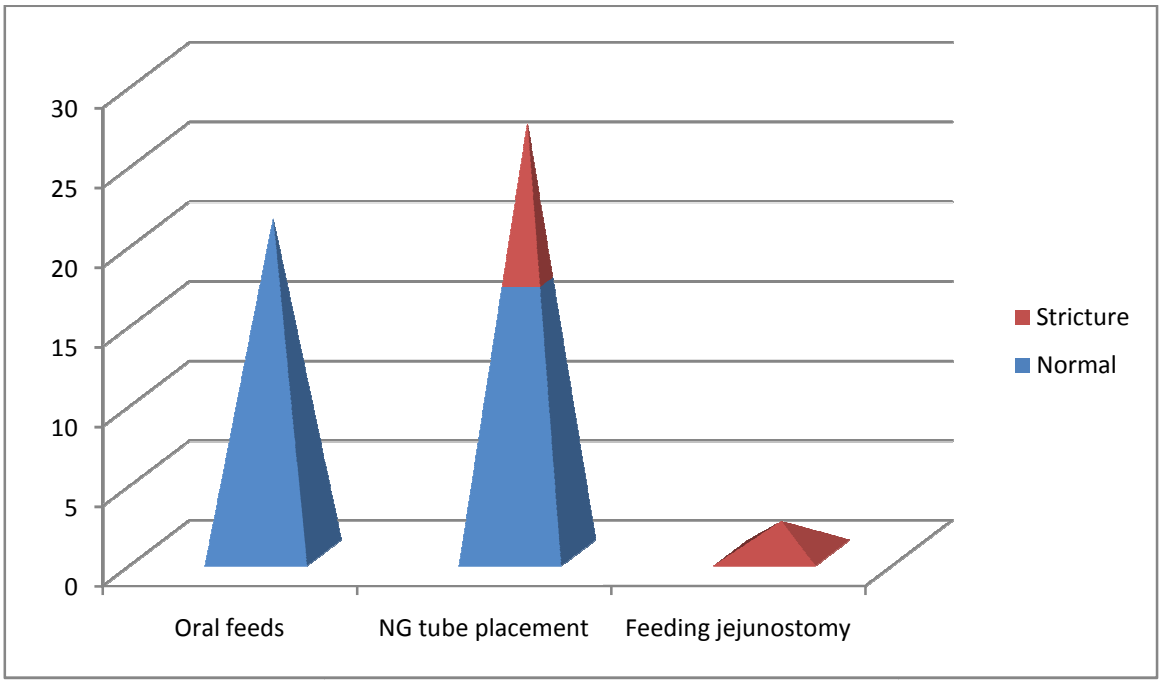


The incidence of stricture was higher in the patients with higher grade of injuries despite the placement of Naso gastric tube and the difference was statistically significant (p value < 0.05).

Table 20 - Analysis of treatment versus outcome:

Feeding procedure	n	I	II
Oral feeds	21	21	0
Naso gastric tube placement	27	10	17
Feeding jejunostomy	2	0	2

Figure 22 – Graph showing treatment given versus outcome.



DISCUSSION

DISCUSSION

Poisoning with corrosive substances is a very common occurrence in our country due to the readily available nature of these compounds in our household products. Ingestion of these substances causes a wide variety of damage to the GI tract both acute (perforation, hemorrhage, etc.) and delayed (stricture, carcinoma). Knowledge about the various predictors of the extent of corrosive is useful in identifying the patients with high risk of complications both acute and delayed. Various Indian studies are available to delineate the epidemiological profile of corrosive consumption in our country but many of them included pediatric corrosive injuries or were done exclusively in pediatric patients.

In our study, observations were made in patients with history of corrosive consumption with respect to the history of consumption, symptoms and signs and findings in initial upper GI endoscopy and they were compared with the final outcome. The following inferences were drawn.

Age distribution

The mean age in our study was 32.88 ±12.74. The youngest age was 15 years and the oldest was 67 years. There was no significant difference in the outcome among different age groups. About 32 percent were in the

age group 20 – 30 years. The mean age in other studies were 26.0 (Zargar et al., Chandigarh (1989)), 21.6 (Lahoti et al., Delhi (1995)) and 26.5 (Gupta and Gupta, Chandigarh (2004)) and a range of 4 – 65 years in the study by Ananthakrishnan et al., Puducherry (2010).

Sex distribution

Among the 50 patients, 36 patients (72%) were males and 14 patients (28%) were females. The male:female ratio was approximately 2.5:1. The higher incidence of corrosive ingestion in males was slightly high but in concordance with other studies where the percentages observed were 66.6% (Gupta and Gupta, Chandigarh (2004)) and 55.0% (Ananthakrishnan et al., Puducherry (2010)). The difference in outcome among males and females was not statistically significant.

Type of corrosive distribution

In our study, ingestion of acids (64%) was more common than alkali ingestion (36%). The occurrence of acid ingestion in our study was slightly lower than other Indian studies where the incidences were higher 83.4% (Gupta and Gupta, Chandigarh (2004)) and 82.6% (Ananthakrishnan et al., Puducherry (2010)). This is a parameter where Indian data differs strikingly from western data where alkali consumption is more common than acid

ingestion. The reason is attributed to the easy availability of acids when compared to alkalis[28]. The most common acid encountered in our study was hydrochloric acid (toilet cleaning acid) followed by phenol (carbolic acid). The common alkali encountered in our study was sodium hypochlorite (bleaching powder) followed by benzalkonium chloride (toilet bowl cleaners).

Circumstance of consumption

In our study suicidal consumption was the most common cause for corrosive ingestion accounting for 76% of cases. Accidental consumption under the influence of alcohol formed 8% of the cases. In India due to the under reporting of attempted suicides, the exact incidence of suicidal consumption of corrosives is not known. The studies done in cases presenting with history of corrosive consumption in tertiary care centres have revealed the following percentages as suicidal consumption - 54.2% (Rao et al., Puducherry (1988)) and 39.0% (Zargar et al., Chandigarh (1989)).

The outcome was not statistically significant different between the patients with suicidal and accidental consumption especially due to the persons with accidental consumption under the influence of alcohol. The severity of injury was also high in the patients with accidental consumption under the

influence of alcohol. In other patients with accidental consumption, those who had ingested amount > 50 ml had higher grading of injury. Occurrence of oropharyngeal burns was higher in the patients with suicidal intention possibly due to the hesitant sipping[29]. This observation has been made by Ananthakrishnan et al., Puducherry (2013).

Duration since consumption:

The mean duration since consumption when the patient was subjected to upper GI endoscopy was 14.06 ± 3.48 hours. The least duration was 8 hours and the maximum duration was 20 hours. The incidence of strictures was higher in the patients who underwent endoscopy later than 12 hours but the difference was not statistically significant. This was probably due to the underestimating of injuries in patients in whom endoscopy was done < 12 hours. Studies have shown that optimal timing for the performance of upper GI endoscopy in post corrosive injuries to be in the first 12 – 24 hours (Andon Chibishev, et al., *The Role of Urgent Esophagogastroduodenoscopy in Prognosis of Acute Caustic Poisonings*, AIM, 2011)[1].

Symptomatology at presentation

In our study, majority of the patients was asymptomatic (78%). The most common clinical features among the symptomatic group were chest pain and dysphagia (12% each). The incidence of stricture was more common in the asymptomatic group but the difference was not statistically significant. Zargar et al in a study of 41 patients noted that dysphagia and odynophagia to be correlated with esophageal injury but epigastric pain and tenderness was not associated with gastric injury at endoscopy[30].

Physical signs at presentation:

Our study of 50 patients with history of corrosive ingestion revealed that oropharyngeal burns were present in only 10% of the patients at the time of presentation. The incidence of significant esophageal and gastric injury as well as stricture was higher in the patients with normal physical examination and the difference was statistically significant. This correlates with studies which have the shown the poor predictive nature of oropharyngheal in predicting the occurrence of esophageal or gastric injury following corrosive ingestion (Previtera et al 1990 done in pediatric population[8], Zargar et al 1991[4]).

Spectrum of injury to the GI tract:

In our study the spectrum of injury was as follows;

The most common pattern observed in the esophagus was II b (32%) whereas in the stomach, the most common pattern was grade 0 injury (22%) followed by grade II b and III a(20% each). The duodenum was spared in majority of the cases (82%) but could not be entered in 6% cases due to extensive gastric injury. Cibisev et al in 2007 observed in their study that the most common pattern of injury was II a (36%) followed by grade II b (25%) and grade III (23%). The once observed dictum that acid spares the esophagus and affects the stomach was observed only in 5 patients with acid ingestion in our study[28].

Outcome of ingestion:

The follow up endoscopy revealed that grade 0, I and II a injuries healed without sequelae while strictures in 19 patients (38%) in patients with grade II b and III a. 17 patients had esophageal strictures in whom further endoscopy was not done. In the remaining patients 2 patients had pyloric strictures. The incidence of esophageal strictures was higher in patients with grade II b and III a injuries (65%) especially with circumferential injuries. This difference was statistically significant. Zargar et al made a

similar observation in1991 where grade 0, I and II a injuries recovered fully while II b and III a lesions went on to develop strictures in as much as 71% of those patients who had esophageal lesions. The placement of NG tube was also associated with the development of strictures in majority of patients hence was not protective in preventing strictures but are nevertheless indicated for maintain nutrition in the acute stages. 4% patients required feeding jejunostomy for nutrition.

LIMITATIONS

LIMITATIONS OF THE STUDY

- Size Of the sample is small.
- Patients with other grave complications such as perforation, respiratory distress were not included in the study and hence outcome in that subset could not be analysed.
- Further follow up and the requirement of further treatment in the form of repeated dilatations or surgical procedure was not analysed.

CONCLUSION

CONCLUSION AND SUMMARY

Our study included 50 patients with history of corrosive ingestion and with endoscopic evidence of corrosive injury. They underwent detailed history elicitation and thorough physical examination and were subjected to Upper GI endoscopy within 24 hours of consumption.

Corrosive ingestion was more common in the age group 20 – 30 years and more common in males. Acid ingestion was almost twice as common as alkali ingestion. Suicidal ingestion was the most common circumstance of consumption and associated with higher grade of injury though accidental intake under the influence of alcohol also had the risk of higher grades of injury and long term sequelae.

Patients with ingestion of more than 50 ml had higher grades of injury and also were at higher risk of strictures. Majority of the patients were asymptomatic. Chest pain and dysphagia were the most common symptoms among the symptomatic patients. Oropharyngeal burns were present only in 10% of patients. Symptoms and physical signs were not reliable in predicting the outcome of injury both acute and long term.

The spectrum of injury to the GI tract revealed esophageal injury of grade II b and gastric injury of grade 0 to be the most common finding with the

duodenum being spared in majority of the cases. While the lesser grade injuries (0, I, II a) were associated with complete recovery with no sequelae, the more severe grades (II b and III a) were associated with higher incidence of strictures especially the circumferential lesions. In our study only one patient with grade III a and 9 patients with grade II b injuries recovered completely. All patients with circumferential lesions went onto develop strictures. Hence the extent of initial GI tract injury at endoscopy had the most significant correlation with the development of strictures later.

The placement of NG tube was not associated with decrease in the occurrence of strictures. Two patients with very severe lesions underwent feeding jejunostomy.

SCOPE FOR FURTHER STUDIES AND RESEARCH

- Evaluation of the usefulness of high dose parenteral Proton pump inhibitors (PPIs) in reducing the severity of injury and decreasing the incidence of strictures.
- Evaluation of the use of endosonography in assessing the extent of corrosive injury of the GI tract.
- Evaluation of the usefulness of non invasive imaging such as CT in predicting the maximal wall thickness and thereby assessing the response of strictures to dilatation and the no. of sessions needed or to decide for surgical modalities.

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ANNEXURES

PROFORMA.

CLINICAL AND ENDOSCOPIC PREDICTORS OF THE OUTCOME OF CORROSIVE INGESTION

- ***NAME :*** ***AGE:***
  SEX:
- ***ADDRESS:*** ***I.P.NO:***

- ***PAST HISTORY:***
  - ***SHT.*** ***DM.*** ***CLD.*** ***CAD.***
- ***DRUG HISTORY:***
  - ***Antiplatelets.*** ***Analgesics***
- ***PERSONAL HISTORY:***
  - ***Smoking.*** ***Alcohol.***
- ***CLINCAL FEATURES:***
  - ***Symptoms:***
    - ***consumption related***
      - ***nature of compound***
      - ***time since consumption***
      - ***quantity of consumption***
      - ***food ingestion prior to consumption (y/n)***
      - ***suicidal or accidental***
    - ***symptoms***
      - ***vomiting***
      - ***hematemesis***
      - ***chest pain***
      - ***shortness of breath***
      - ***abdominal pain***
  - ***Signs:***
    - ***Vitals***

      PR: ***BP:*** ***RR:*** ***TEMP***

- ***EXAMINATION OF THE ABDOMEN***
  - ***Tenderness*** ***Warmth***
  - ***Organomegaly*** ***Free fluid***
- ***OTHER SYSTEMS :***

CVS; ***RS;***

CNS;

- ***INVESTIGATIONS:***
  - ***CBC:*** ***RFT:*** ***LFT:***

- ***ECG***
- ***X ray Chest PA view***
- ***X ray Abdomen erect***
- ***USG abdomen and pelvis***
- ***OGD scopy***

	At admission	***At 6 weeks***
Oral cavity		
Oesophagus		
Stomach		
Duodenum		

INFORMATION SHEET

We are conducting a study on **"CLINICAL AND ENDOSCOPIC PREDICTORS OF THE OUTCOME OF CORROSIVE INGESTION"** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us.

The purpose of this study is to assess the outcome of corrosive ingestion in patients within 24 hrs of consumption.

We are selecting certain cases and if you are found eligible, we may be using your blood samples to do special studies which in any way do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator | Signature of Participant

Date :

Place :

PATIENT CONSENT FORM

Study Detail	:	"CLINICAL AND ENDOSCOPIC PREDICTORS OF THE OUTCOME OF CORROSIVE INGESTION"
Study Centre	:	Rajiv Gandhi Government General Hospital, Chennai.
Patient's Name	:	
Patient's Age	:	
Identification Number	:	

Patient may check (√) these boxes

a) I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ❑

b) I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ❑

c) I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ❑

d) I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ❑

e) I hereby consent to participate in this study. ❑

f) I hereby give permission to undergo complete clinical examination and hematological tests and OGD scopy. ❑

Signature/thumb impression	Signature of Investigator
Patient's Name and Address:	Study Investigator's Name: **Dr. KARTIKAYAN. R. K.**

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

EC RegNo.ECR/270/Inst./TN/2013
Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr.R.K.Kartikayan,
PG in MD General medicine
Madras Medical College, Chennai-3.

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Dear Dr.R.K.Kartikayan

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Clinical and endoscopic predictors of the outcome of corrosive ingestion" No.28072013.

The following members of Ethics Committee were present in the meeting held on 02.07.2013 conducted at Madras Medical College, Chennai -3.

1. . Dr.G.SivaKumar, MS FICS FAIS --- Chairperson
2. Prof. R. Nandhini MD -- Member Secretary
   Director, Instt. of Pharmacology ,MMC, Ch-3
3. Prof. Shyamraj MD -- Member
   Director i/c , Instt. of Biochemistry , MMC, Ch-3
4. Prof. P. Karkuzhali. MD -- Member
   Prof., Instt. of Pathology, MMC, Ch-3
5. Prof. Kalai Selvi -- Member
   Prof of Pharmacology, MMC, Ch-3
6. Prof. Siva Subramanian, -- Member
   Director, Instt. of Internal Medicine, MMC, Ch-3
7. Thiru. S. Govindsamy. BABL -- Lawyer
8. Tmt. Arnold Saulina MA MSW -- Social Scientist

We approve the proposal to be conducted in its presented form.

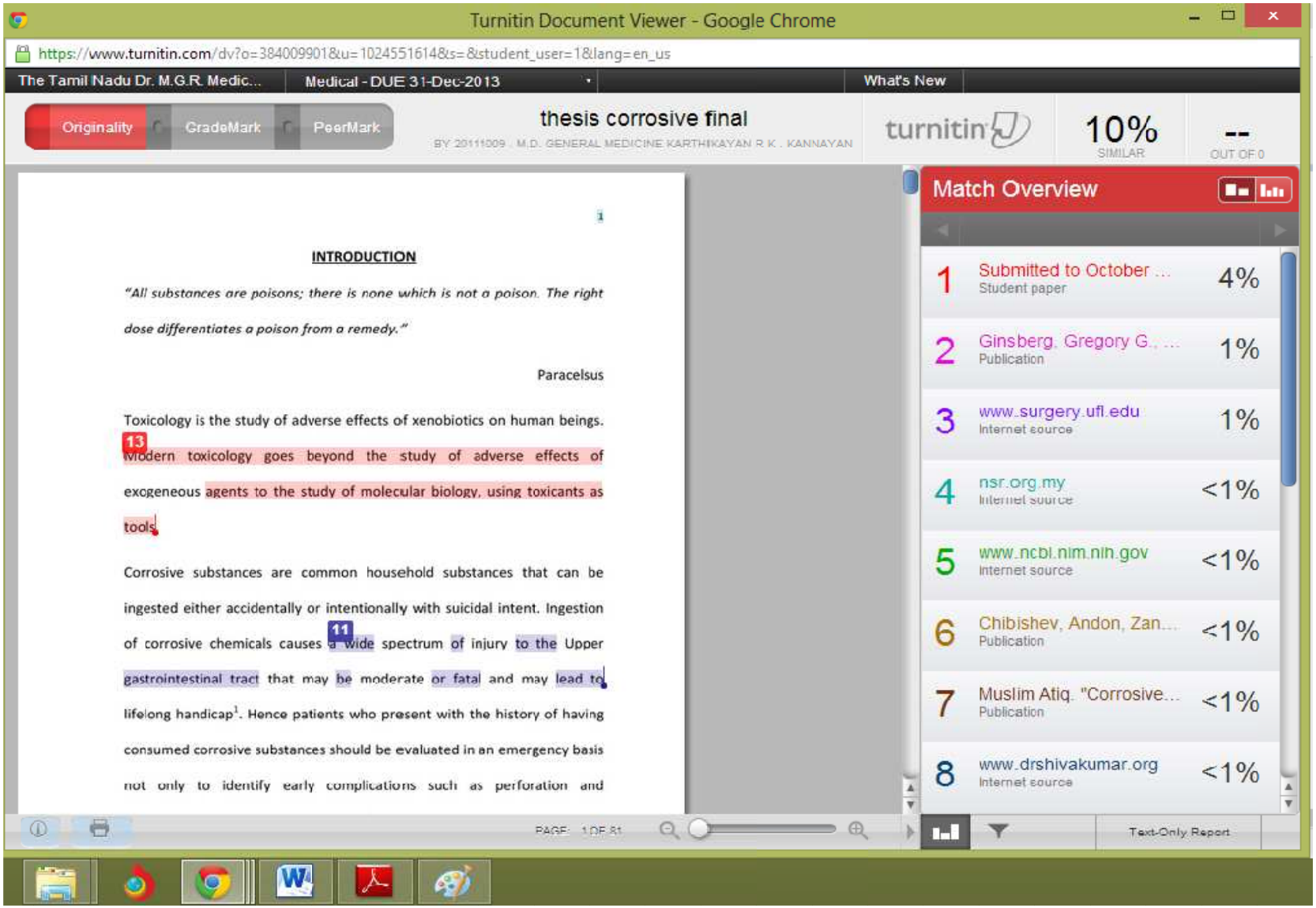
Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nadini 12/7/13

Member Secretary, Ethics Committee

S. No	age	sex	amount cons	acid/alkali	suicidal/accidental	duration	symptomatology	physical signs	scopy - esophagus	stomach	duodenum	treatment	follow up scopy
1	35	male	< 50 ml	acid	suicidal	10 hrs	nil	nil	II a	normal	normal	oral feeds	normal
2	20	male	< 50 ml	alkali	suicidal	9 hrs	nil	nil	normal	I	normal	oral feeds	normal
3	30	male	< 50 ml	acid	suicidal	14 hrs	nil	nil	II a	normal	normal	oral feeds	normal
4	50	male	< 50 ml	alkali	suicidal	12 hrs	nil	nil	II b circumferential	II b	normal	NG tube placement	stricture at 20 cms
5	45	female	< 50 ml	acid	suicidal	10 hrs	nil	nil	II b	II a	normal	NG tube placement	normal
6	25	female	< 50 ml	alkali	suicidal	10 hrs	nil	nil	I	I	normal	oral feeds	normal
7	43	female	> 50 ml	acid	suicidal	14 hrs	nil	nil	II b	III a	II a	NG tube placement	stricture at 19 cm
8	33	male	< 50 ml	alkali	suicidal	20 hrs	nil	nil	II a	I	normal	oral feeds	normal
9	16	female	> 50 ml	alkali	suicidal	18 hrs	chest pain, dysphagia	oropharyngeal burns	III a	III a	not entered	feeding jejunostomy	stricture at 18 cms
10	57	male	< 50 ml	acid	accidental	14 hrs	nil	nil	II a	normal	not entered	oral feeds	normal
11	32	male	< 50 ml	acid	accidental	18 hrs	chest pain, vomiting	nil	I	normal	normal	oral feeds	normal
12	36	male	> 50 ml	alkali	suicidal	12 hrs	haemetemesis	nil	II b	normal	normal	NG tube placement	normal
13	50	male	not known	acid	suicidal	10 hrs	nil	nil	II b	III a	normal	NG tube placement	normal
14	35	male	< 50 ml	acid	suicidal	18 hrs	nil	nil	III a	II b	normal	NG tube placement	stricture at 18 cm
15	21	male	> 50 ml	acid	accidental	15 hrs	nil	nil	normal	II a	normal	oral feeds	normal
16	56	female	> 50 ml	acid	suicidal	16 hrs	chest pain, vomiting	nil	III a circumferential	II b	normal	NG tube placement	stricture at 20 cms
17	26	male	< 50 ml	alkali	suicidal	14 hrs	nil	oropharyngeal burns	III a circumferential	II b	normal	NG tube placement	stricture at 24 cms
18	25	male	> 50 ml	acid	suicidal	9 hrs	nil	nil	III a	II b	not entered	NG tube placement	stricture at 15 cms
19	23	male	< 50 ml	acid	accidental	10 hrs	nil	nil	normal	I	normal	oral feeds	normal
20	43	male	not known	acid	accidental under alcohol in	9 hrs	nil	nil	III a	III a	erosions	NG tube placement	stricture at 28 cms
21	43	male	> 50 ml	acid	suicidal	10 hrs	chest pain	nil	III a circumferential	III a	not entered	NG tube placement	Pylorus distorted - GOO
22	26	male	< 50 ml	alkali	accidental	12 hrs	nil	nil	I	normal	normal	oral feeds	normal
23	36	male	> 50 ml	acid	suicidal	20 hrs	nil	nil	II b circumferential	normal	normal	NG tube placement	stricture at 10 cm
24	22	male	> 50 ml	acid	suicidal	16 hrs	nil	nil	III a	III a	normal	NG tube placement	stricture at 20 cms
25	32	male	not known	acid	accidental under alcohol in	17 hrs	dysphagia, vomiting	oropharyngeal burns	II b	II b	II b	NG tube placement	partial pyloric stricture
26	47	male	> 50 ml	acid	suicidal	15 hrs	chest pain, vomiting	nil	II b	II b	normal	NG tube placement	stricture at 26 cms
27	17	female	not known	alkali	suicidal	16 hrs	nil	oropharyngeal burns	II b	III a	normal	NG tube placement	normal
28	17	female	> 50 ml	alkali	suicidal	12 hrs	nil	nil	II a	II b	normal	NG tube placement	normal
29	36	male	> 50 ml	acid	suicidal	12 hrs	vomiting,	nil	III a	II a	normal	NG tube placement	stricture at 24 cms
30	26	male	< 50 ml	alkali	suicidal	20 hrs	nil	nil	II b	normal	normal	NG tube placement	normal
31	33	male	not known	acid	accidental under alcohol in	15 hrs	nil	nil	normal	II a	I	oral feeds	normal
32	28	male	< 50 ml	acid	suicidal	10 hrs	nil	nil	I	normal	normal	oral feeds	normal
33	38	male	> 50 ml	acid	suicidal	12 hrs	chest pain, vomiting	oropharyngeal burns	III a	III a	II a	feeding jejunostomy	stricture at 28 cms
34	25	male	> 50 ml	acid	suicidal	12 hrs	nil	nil	III a	II a	normal	NG tube placement	normal
35	19	male	> 50 ml	acid	accidental	17 hrs	nil	nil	II b	III a	normal	NG tube placement	normal
36	19	male	> 50 ml	acid	suicidal	12 hrs	nil	nil	II b	II a	normal	NG tube placement	normal
37	22	male	< 50 ml	alkali	suicidal	10 hrs	nil	nil	II a	normal	normal	oral feeds	normal
38	28	male	< 50 ml	acid	suicidal	14 hrs	nil	nil	II a	normal	normal	oral feeds	normal
39	55	female	< 50 ml	alkali	suicidal	16 hrs	nil	nil	II b	II b	normal	NG tube placement	stricture at 18 cms
40	30	female	< 50 ml	acid	suicidal	14 hrs	dysphagia	nil	II b	II b	normal	NG tube placement	stricture at 26 cms
41	15	female	< 50 ml	alkali	suicidal	15 hrs	nil	nil	I	I	normal	oral feeds	normal
42	23	female	> 50 ml	acid	suicidal	20 hrs	vomiting, chest pain	nil	II a	II a	normal	oral feeds	normal
43	35	female	< 50 ml	alkali	suicidal	14 hrs	nil	nil	II b	II b	normal	NG tube placement	stricture at 25 cms
44	57	male	< 50 ml	acid	suicidal	20 hrs	nil	nil	normal	I	I	oral feeds	normal
45	67	male	< 50 ml	acid	accidental	18 hrs	nil	nil	II b	I	normal	oral feeds	normal
46	22	female	> 50 ml	alkali	suicidal	13 hrs	nil	nil	III a circumferential	II a	normal	NG tube placement	stricture at 28 cms
47	45	male	< 50 ml	alkali	accidental	18 hrs	nil	nil	II a	I	normal	oral feeds	normal
48	23	male	< 50 ml	alkali	suicidal	14 hrs	nil	nil	I	I	normal	oral feeds	normal
49	17	female	< 50 ml	acid	suicidal	19 hrs	nil	nil	I	I	normal	oral feeds	normal
50	40	male	not known	acid	accidental under alcohol in	8 hrs	nil	nil	II a	III a	normal	NG tube placement	normal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INTRODUCTION
"All substances are poisons; there is none which is not a poison. The right dose differentiates a poison from a remedy."
Paracelsus
Toxicology is the study of adverse effects of xenobiotics on human beings. Modern toxicology goes beyond the study of adverse effects of exogeneous agents to the study of molecular biology, using toxicants as tools
Corrosive substances are common household substances that can be ingested either accidentally or intentionally with suicidal intent. Ingestion of corrosive chemicals causes a wide spectrum of injury to the Upper gastrointestinal tract that may be moderate or fatal and may lead to lifelong handicap[1]. Hence patients who present with the history of having consumed corrosive substances should be evaluated in an emergency basis not only to identify early complications such as perforation and
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1 INTRODUCTION “All substances are poisons; there is none which is not a poison. The right dose differentiates a poison from a remedy.” Paracelsus Toxicology is the study of adverse effects of xenobiotics on human beings. Modern toxicology goes beyond the study of adverse effects of exogeneous agents to the study of molecular biology, using toxicants as tools. Corrosive substances are common household substances that can be ingested either accidentally or intentionally with suicidal intent. Ingestion of corrosive chemicals causes a wide spectrum of injury to the Upper gastrointestinal tract that may be moderate or fatal and may lead to lifelong handicap1. Hence patients who present with the...